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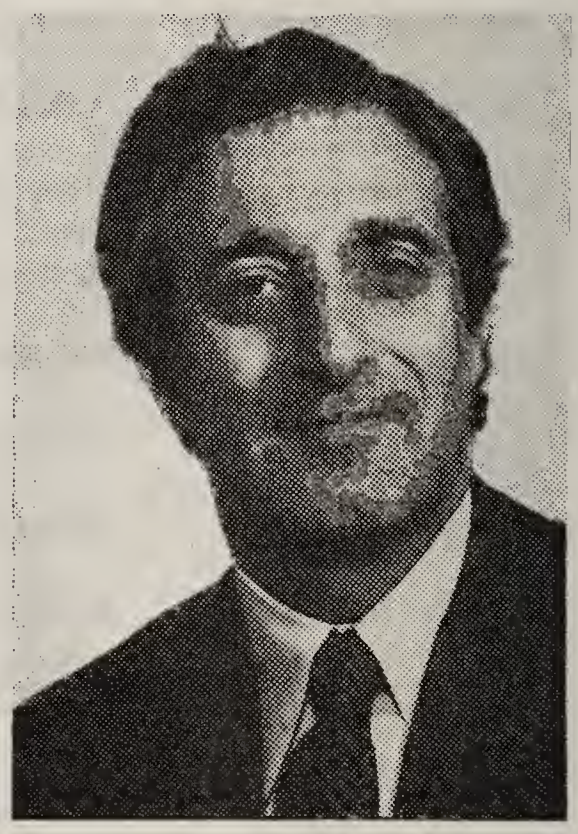
GUIDELINES FOR PUBLICATION

1) All articles intended for publication should be submitted to the editors before the deadline which is usually six weeks before the release of the Journal.
2) Submitted articles should be typed (or legibly printed) double-spaced, single-sided on paper 8 1/2" x 11" (letter size) and they should not be more than 15 to 20 pages (3000 to 4000 words).
3) Illustrations, graphs, diagrams and figures should be put on a separate sheet and identified by Arabic numerals. A list of explanatory legends for all the figures and diagrams should be put on another sheet. Do not write on the back of photographs.
4) References should be checked with the original sources, designated by Arabic numerals in the text arranged in Alphabetical order and put on a separate sheet. Each periodical reference should include in order the author's name and initials title of the article, Journal name, volume number, page number and year (eg. 1 Rosen HJ Lumbar Invertebral disc surgery, review of 300 cases *Can Med Assoc J. 101 317, 1969)*.
5) The editors reserve the right to make such editorial changes as are deemed necessary to ensure correctness of grammar and spelling, clarification of statements and conformity to Journal style.
6) Acceptance for publication is subject to the understanding that such material is submitted solely to the Jornal and will not be reprinted without the consent of both the editors and author.

The Dean's Challenge

Frederick H. Lowy, B.A., M.D., C.M., C.R.C.P.(C), F.R.C.P.(C)



Dr. Frederick H. Lowy was born in Grosspetersdorf, Austria, and attended McGill University, where he received his B.A. in 1955 and M.D., C.M. in 1959. After finishing his postgraduate residency in Psychiatry at Cincinnati General Hospital and Veterans' Administration Hospital, Cincinnati, Ohio in 1965, he furthered his training in Psychoanalysis at Canadian Institute of Psychoanalysis, Montreal until 1970. From 1974 to 1980, he held positions as Professor and Chairman of the Department of Psychiatry at the University of Toronto, as well as Director and Psychiatrist-in-chief at Clarke Institute of Psychiatry. Since 1980, Dr. Lowy has been named Dean, Faculty of Medicine, University of Toronto.

In addition to Dr. Lowy's appointment as Honorary Consultant of various hospitals in Ontario, including the Ontario Cancer Institute and Addiction Research Foundation, he is also an active member of numerous Societies and Committees. For the past few years, he has been the Corresponding Editor of the International Journal of Psychiatry in Medicine and Head of the Psychotherapy Research Section at Clarke Institute of Psychiatry. Dr. Lowy has published over 40 articles and reviews, and has been a contributor to several major text books, including Greben's A Method of Psychiatry *and Salvendy's* Psychiatric Out-Patient Treatment in the 80's.

I very much welcome the editors' invitation to use the pages of the Medical Journal as another forum for dialogue with the student body. Given this opportunity I wish to address a question that to-date only students have asked me. It is one that I first found surprising, and now recognize to be as profound and fundamental as it is direct and simple: "What", I have been asked by several students, "does a Dean do?"

Indeed, students may well wonder what, if anything, the Dean does between the ceremonial occasions when he appears to welcome them to the Faculty, introduce symposia and lectures, or confer degrees at graduation. As I thought about this it became evident that a proper answer goes beyond my specific job description as Dean to a consideration of the challenges that confront me and this Faculty of Medicine in the early 1980's.

I will therefore try to provide not only an overview of my tasks and responsibilities but also to share with you my perception of some of the problems that we must attempt to solve.

The Dean's Job

A review of the Dean's official position description reveals that my duties can be summarized under three classes of responsibilities. The Dean is responsible to:

A. The University

1. Responsible to the Provost, through the Vice-Provost, Health Sciences, for the Faculty's academic programs and the management of the resources (budget, space personnel).

2. Responsible for ensuring that University policies, regulations and procedures are adhered to within the Faculty.

3. Responsible for the Faculty's representation on University Councils, committees etc.

B. The Faculty

1. Responsible to Faculty Council for carrying out the academic policies it determines and the objectives it sets.

2. Responsible for strategic and tactical planning; that is, how the Faculty's present and projected resources can best be used to achieve changing academic and clinical objectives.

3. Responsible for recommending the appointment of Faculty Officers and Department

Chairmen (on the advice of search committees) and, with them, translating Faculty goals into specific departmental objectives.

C. The Health Care System

1. Responsible for ensuring effective liaison between the Faculty and its affiliated teaching hospitals to provide the clinical milieux wherein optimal clinical teaching can occur.

2. Responsible for ensuring that the Faculty plays appropriate roles *vis-a-vis* the health care system in Metropolitan Toronto, Ontario and Canada.

Put very simply, the Dean is the Chief Executive Officer responsible for administering the work of the Faculty; i.e., implementing the academic policy of the institution, as defined by Faculty Council, with the resources provided by the University Administration, the Ministry of Health, public and private research granting agencies etc.

It hardly needs to be stressed, however, that a bureaucratic position description does not capture the essence of most jobs, let alone those that involve people engaged in creative meaningful missions that require at times teamwork, at times individual initiatives. Whatever the official list of duties of, say, a professional football coach or a conductor of a symphony orchestra, the essence of the job lies in the capacity to create circumstances which will assist highly competent professionals to function at their best, and in harmony.

At this medical school, one of the largest in North America, a group of carefully selected, gifted medical students are taught by scientists and clinicans of high national and even international reputation in training facilities that, on the whole are modern and very satisfactory. The network of affiliated institutions includes some of Canada's best teaching general hospitals and three one-of-a-kind specialty institutions, the Clarke Institute of Psychiatry, the Hospital for Sick Children and the Ontario Cancer Institute/Princess Margaret Hospital that are recognized internationally.

The chief role of the Dean is to ensure that the teachers and investigators in these institutions and in the Faculty's campus buildings have the best possible resources, motivation and working climate to enable them to perform their tasks.

At one time even Deans of large medical schools were part-time administrators, borrowing time from their primary duties in the clinics, the wards or the laboratories. Our Associate Deans still divide their time but for the past two decades in Toronto being Dean has been a full time job. If the Dean enjoys teaching and clinical practice (as I do) he will find a way to keep his hand in but his effectiveness will be judged by how well he helps others do their jobs, and on the basis of the outcome - the quality of the education that the students receive, the quality of the science conducted by the staff and the quality of the patient care practiced here, which will be the model for tommorrow's physicians and other health professionals.

The Faculty's Goals

The basic objectives of our medical schools are the same irrespective of size and location:

1. to select and educate students to be competent and ethical health professionals.

2. To select and train competent investigators and scholars in scientific and applied fields.

3. To conduct research and advance knowledge.

4. To serve their communities by providing exemplary patient care in university-affiliated institutions.

5. To provide continuing education to practising health professionals.

However, here at the University of Toronto there are additional considerations. As one of Canada's oldest, its largest and, in many areas, its leading medical school, we have special responsibilities. Fully 40% of Ontario's physicians are trained here, an awesome challenge for the Faculty in that it contributes in a major way to the shaping of tommorrow's health system and the quality of medical care that our citizens will receive. We must provide our students with the information base, the attitudes and the learning skills that will enable them to assimilate present and future advances in knowledge, and then to apply them wisely.

Unlike most other Canadian medical schools, which focus their resources to a greater extent, this Faculty has acquired responsibilities in virtually every area of medicine, its related basic sciences, and the allied health professions. For example we have programs in behavioral science, health administration, speech pathology, physical and occupational therapy, art as applied to medicine, and so on. Members of this faculty have major teaching responsibilities in other faculties, such as arts and sciences, dentistry and pharmacy.

But this Faculty has not only extensive but also intensive academic objectives. The Faculty has in its ranks some of Canada's leading scientists, both basic and applied, who aspire not only to competence but to excellence in research, at the cutting edge of knowledge in their fields. Similarly, our specialized training programs cannot be satisfied merely with competent performance. In most of the medical specialties the mantle of national leadership has clearly fallen on the shoulders of our post-graduate programs.

Tommorrow's academics and professional leaders will emerge from these programs and the only standard of performance that can be acceptable is that of excellence, as judged by world standards. And our community health and clinical divisions should not be satisfied by merely helping existing health care patterns to improve, they must take positions of leadership and innovation in effecting and evaluating model programs of disease prevention, health care and health maintenance.

Unfortunately, even an annual

budget of some $35 million and a cash flow of more than $65 million, counting Faculty administered research grants and endowment income, do not adequately meet the needs of more than 2,000 academics and their myriad teaching and research programs.

Choices have to be made and these must be wise as well as popular; mindful of future as well as present needs; and, above all, sensitive to the interests of the various constituencies served by the Faculty - its students and staff, the University, the medical profession and the health care system and, ultimately, the public.

It will be readily apparent that there must at times be some tension between two worthwhile yet competing goals — preparing large numbers of students for a life of service to the community and stimulating the development of world class performance by a smaller group of investigators and tertiary care specialists. Such tension, probably inevitable even in favourable economic times, is exacerbate by today's budgetary stringency.

There is also the matter predicting and responding to the further evolution of health care in industrial countries. Dr. John Evans, former President of the University, has recently traced this through three stages, each characterized by its own specific problems[1]. The first stage was the broad conquest of the major infectious diseases and the factors (malnutrition, crowding, poor sanitation, etc) that breed these — by public health measures such as immunization and social and economic improvements. This was followed by the control of acute infections by chemotherapy and more advanced immunotechniques delivered in a more personal way, in doctors' offices and clinics.

The successful management of the first stage permitted a shift of attention to chronic diseases, such as cancer, pulmonary, cardiovascular, and cerebrovascular diseases, and the major mental disorders. In this second stage, much of the treatment previously provided in doctors' offices and patients' homes has shifted to hospitals where expensive technology and specialists are available. Highly sophisticated diagnostic and therapeutic tools have become available to assist in the personal, individually oriented care that physicians strive to provide.

At present we are, of course, solidly in this stage of development but, as Evans points out, there is evidence that its peak may have been reached. Many chronic and even degenerative diseases are under control, including the major psychoses, diabetes and a few neoplasms, and there seems to be some reduction in premature death from heart disease.

We are already into a third stage of confronting social and environmental pathology. "Threats to health arise not from intrinsic disorders of structure and function of the body but from environmental exposure and from changes in social conditions — in the family, community and workplace — associated with absenteeism, violence, alcohol and drug abuse of epidemic proportions. The personel health care system only deals with the consequences of such processes[2]." Clearly, new approaches will be required in the face of environmental and occupational hazards, life-style generated disorders and psychosocial pathology that manifests as both illness and illness-behaviour.

To Dr. Evans' analysis I would add that a fourth stage is also virtually upon us, namely, the imminent attack on genetically determined and genetically influenced disorders (and there is a vast array of these) that will be made possible by the startling advances in molecular biology at this very time.

Clearly, it is the Faculty's responsibility to ensure that teaching, learning and research take into account this evolution and that resources (time, personnel, space) be allocated with a view to present and future rather than historical needs.

The Dean must lead (persuade, cajole, browbeat, etc) the faculty to mobilize its analytic and planning capacity to meet these challenges and to effect whatever restructuring is called for.

The Personal Dimension

The administrative and organizational difficulties and the frustrations associated with being a dean have produced a high turnover rate in the United States where the half life of deans was reported by Glaser to be less than three years[3]. Rogers, looking back on three and one half years as Dean at Johns Hopkins University Medical School was moved to declare that "...the deanship in most of our medical schools has become one of the most unattractive professional roles in medical academe[4]".

Perhaps because I am still new in the position after only 18 months, or because there are relative advantages for deans on the Canadian side of the border, I do not at all agree. Quite apart from the distinct honour of becoming the 12th Dean of Medicine in Toronto (the 23rd if one includes the medical schools that preceded and then joined to form this Faculty), there are personal challenges that are as full of opportunity as they have potential for frustration.

Management in an organization comprised of interesting, often brilliant people can be a most creative endeavour. Although the sheer size of our faculty creates many problems it also provides flexibility, but to expand on this point would be beyond the scope of this paper.

Rather, I wish to emphasize the enormous potential gratification for the dean of making a contribution, however small, to the education available to our students and thereby to the public, the ultimate consumers of health care and research advances. Such a contribution will more often be made indirectly, by favourably in-

fluencing the Faculty infrastructure that supports teaching, research and exemplary care, but at times directly through contact with students and teachers.

Again, specific consideration of the dean's potential contributions and the particular changes that I wish to foster will require a separate paper.

There is no doubt that changes can and should be made to improve further on teaching methods, the curriculum itself, the emotional climate in which students learn and prepare for examinations, the other stresses that impringe on students, and so on.

It is a task both challenging and welcome.□

References

1. Evans, J.R.: *Measurement and Management in Medicine and Health Services*, New York: The Rockefeller Foundation, 1981
2. Ibid.
3. Glaser, R.J.: The Medical Deanship: Its Half Life and Hard Times, *J. Med EJDUC.* 44: 1115, 1969.
4. Rogers, D.E.: The PHAROS *(AOA)*, p. 115, July 1975.

MEDICAL ETHICS ESSAY CONTEST

Students are invited to submit original essays on topics of their choice.

DEADLINE: March 1st, 1982
LENGTH: max. 4000 words
SUBMIT TO: the editor,
Medical Journal,
med. soc. office

PRIZES: $50, 30, 20

Winning entries will be published in the Medical Journal.

Endorphins

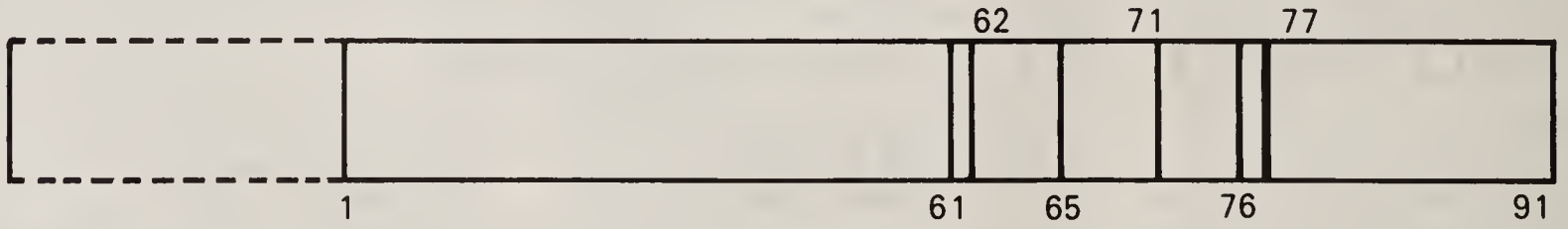
Richard Cheng Ph.D. 8T3

The detection of the opiate receptors aroused the search for endogenous opiates. It was predicted that opiate receptors and the "endogenous opiates" served as a lock-and-key system. The hypothesis was: since there is a lock, there must be a key to fit this lock. Successful results were obtained by several groups of workers; several endogenous ligands of the opiate receptors were detected in the brain, pituitary, CSF and peripheral tissues.

Types of Endorphins

Different types of endorphins have been detected in the pituitary, brain spinal cord, CSF, blood plasma and peripheral tissues. The following lists the types of endorphins that have been detected:

1. Methionine-enkephalin (H-Tyr-Gly-Gly-Phe-Met-OH)
2. Leucine-enkephalin (H-Tyr-Gly-Gly-Phe-Len-OH)
3. Pro-opiocortin 31,000 or 37,000 daltons
   (Precursor for ACTH and beta-LPH)



Beta-Lipotropin (1-91)
Beta-endorphin (61-91)
Alpha-endorphin (61-76)
Gamma-endorphin (61-77)
Des-tyrl-gamma-endorphin (62-71)
Met-enkephalin (61-65) — (Fraction II)

4. Morphine-like compound (non-peptide)
5. Beta-Leu 5-endorphin (31 amino acids)
6. Dynorphin (17 amino-acids)
7. Fraction I (CSF), Plasma Factor (Plasma), unidentified brain opioid peptide (brain)
8. Hexapeptides and Heptapeptides (unnamed)
9. Alpha-neo-endorphin (12 amino acids)

Other related substances
1. Endorphin releasing factor (H-Tyr-Arg-OH)
2. Endogenous anti-opiate substance

Location of Endorphins

a) Enkephalins: Met-enkephalins and leu-enkepheilins appear to be neurotransmitters in nerve terminals and mimic the effect of morphine by binding to opiate receptors. They are found mostly in the brain and spinal cord.

b) B-endorphin: This is made and relased together with ACTH in the hypothalmus and anterior pituiary.

c) Dynorphin: Recently, Goldstein and colleagues (1979) have successfully identified a novel pituitary endorphin dynorphin. It is a tridecapeptide containing (Leu)-enkephalin and is 700 times more potent than leu-enkephalin in inhibiting the guinea pig ileum longitudinal muscle contraction. This effect is only partially reversed by naloxone.

Function of Endorphins

In general, endogenous opioid peptides act similarly to morphine which has a profound effect on the bodily functions including physiological and behavioral in human and animals. There are different types of opiate receptors and endorphins which are located in numerous areas of the CNS and other parts of the body. The functions of endorphins are numerous and variable. The physiological or behavioral variability and characteristics of endorphin actions depend on the location, action and types of endorphin-opiate receptor activities. In the past few years, met-enkephalin, leu-enkephalin, beta-enkephalin and their analogues as well as other endorphins have been extensively studied. Their effects on animals can be summarized in the following list (to be amplified later in the text):

a. Analgesia: relieve pain and modulate stress

b. Homeostasis:

(i) Releasing regulator of other hormones

Prolactin ↑, LH ↓

Growth hormone ↑, FSH ↓

Thyrotrophin ↑

Corticosterone synthesis ↑

release oxytocin and vasopressin

(Beta-endorphin and ACTH secreted concomitantly)

(ii) Thermoregulation

Hypothermia (Alpha-endorphin)

Hyperthermia (Beta-endorphin)

(iii) Digestive system. Inhibit pancreatic secretions and increase insulin and glucagon release. Suppress intestinal motility in small intestine.

(iv) Cardiovascular system. Depress heart and blood pressure.

(v) Respiratory system - Depress respiration.

c Mental illness:

(i) Schizophrenia (abnormal high level of CSF endorphins, Leu (5)-beta-endorphin found in kidney dialysis).

(ii) Small dose of beta-endorphin causes catatonia (akinesis), epilepsy and limbic seizures.

(iii) High doses of naloxone reduces hallucination cf schizophrenic patients.

(iv) des-Tyr-gamma-endorphin - a neuroleptic substance.

d. Behaviour:

(i) Interact with other neurotransmitters - acetylcholine, dopamine, GABA, 5HT, noradrenaline

(ii) Maintain normal behaviour -

alpha-endorphin (tranquilization)

gamma endorphin (agitation and violent behavior)

beta-endorphin (catatonia)

Morphine and Endorphins (euphoria)

Leu-enkephalin (Reward-Pleasure-Learning)

Abnormally high pituitary beta-endorphin (Obesity)

des-tyr-gamma-endorphin (tranquilizer)

(iii) Sexual regulation -

High dose (6 μg) suppressed copulatory behavior of rats.

Low dose (3 ug) increased mounting and intromission latencies.

(iv) Naloxone was found to enhance memory and morphine antagonized this effect (Messing et al, 1979)

e. Addiction: - Morphine and endorphin are addictive.

a) Analgesia

It has been verified that endorphins mimic the effect of opiates. Opiate receptors appear most densely in the paleospinothalamic pathway that transmits diffuse pain, and application of endorphins either in the CSF or in various nociceptive centres of the brain causes analgesia. Presently Pert and colleagues (1979) have reported that Type I opiate receptors are located in the brain areas that mediate analgesia. Type I receptors are probably responsible for mediating endogenous pain-relief while Type II opiate receptors are responsible for the feeling of sedation, dysphoric and hallucinatory syndromes. Experimental evidence indicates that analgesia is observed after microinjection of met-enkephalin (120 ug/injection) into or near the ventral, caudal midbrain, and PAG (Belluzzi et al, 1976; Bradley et al, 1976; Buscher et al, 1976: Frederickson and Norris, 1976), while seizures and other pathological EEG changes are seen with injections into or near the forebrain dorsomedial nucleus of the thalamus (Frenk et al, 1978). The noxious response of thalamic nociceptive neurons is also depressed by systemic or iontophoretic injections of D-ala (2) or D-Leu (5)-enkephalins (Hill and Pepper, 1978). Met-enkephalin (0.2 - 20 ug/rat) and leu-enkephalin (1-20 ug/rat) produced a dose-related and naloxone antagonizable analgesia when microinjected into the nucleus reticularis gigantocellularis and nucleus reticularis paragigantocellularis of the medulla ablongata (Takagi et al, 1978). Microiontophoretical application of met-enkephalin into laminae I, II and III selectively suppresses the excitability of nociceptive dorsal horn neurons located in laminae V (Duggan et al, 1977).

Enkephalin analgesia is one type of the endogenous pain-control system which functions at the brainstem and spinal cord levels. Met-enkephalin acts like neurotransmitters that are released through the synapses of nerve terminals (Snyder, 1977). Enkephalin-degradating enzymes (enkephalinase) and opiate receptors have markedly heterogenous and parallel distribution throughout the mouse brain (Malfroy et al, 1979). Extremely rapid degradation of met-enkephalin is observed in vivo and vitro (Dupont et al, 1977). Thus a enkephalin pain-control system tends to be localized at the segmental or regional areas of the body and the analgesic effect will be short-lasting.

The total brain enkephalin levels (by radioimmunoassay, RIA) represent only 2-13% of the total endorphins radioreceptor assay (RRA) (Gros et al, 1978; Llorens-Cortes et al, 1977). Perhaps the other endorphins may play a role for more profound, generalized and long-lasting pain-relief (e.g. beta-

endorphin and dynorphin).

Evidence indicates that intraventricular (Moroni et al, 1978; Hosobuchi and Li, 1978) and intravenous (Loh et al, 1976) administration of beta-endorphin can induce analgesia in human and animals. Stimulation-produced analgesia by implanting electrodes at PAG (Hosobuchi et al, 1979) or periventricular brain areas (Akil et al, 1978) increases betaendorphin in human ventricular CSF by 50 to 300%, while stimulating the posterior limb of the internal capsule has no such effect (Hosobuchi et al, 1979). Foot-shock induced analgesia is reversed by naloxone and is cross-tolerant with morphine in mice (Chesher and Chan, 1977). However this is in contrast to the results observed by Akil et al (1978), in which stress-induced analgesia in rats shows no cross-tolerance to morphine. This foot-shock induced stress produces naloxone-reversible hyperthermia (Blasic et al, 1978) and increases plasma beta-endorphin levels six fold (Rossier et al, 1977). B-LPH, B-MSH and B-endorphin can pass from the circulation to the CSF and circulating B-LPH can be cleaved to beta-endorphin (Pezalla et al, 1978). Beta-endorphin has a half-life of 9.3 ± 0.75 minutes (Chang et al, 1978), but the pain-relief induced by systemic injection of beta-endorphin will be long-lasting and throughout the whole body.

Naloxone reverses endotoxin hypotension (Holaday and Faden, 1978) and has great potential therapeutic value for shock (like severe blood loss) (Faden and Holaday, 1979). These results suggest that endorphins play a role in shock but the true mechanism still has to be explored.

Recently synthetic analogues of enkephalins which have been made by changing the amino acid sequence can resist enzymatic degradation and can pass through the blood-brain barrier. Thus they can be taken orally to cause analgesia and their analgesic potency can be tremendously increased (even up to 30,000 fold as compared to met-enkephalin) Snyder, 1977). Unfortunately they are highly addictive drugs.

b) Homeostasis

High concentrations of endorphins in the hypothalamus and adenohypophysis suggest that they play a role in pituitary endocrinological functions. Enkephalin neurons project from the hypothalamus to the pars nervosa in the pituitary and may be involved in the regulation of neurohypophyseal neurosecretion (Rossier et al, 1979). Enkephalins (May et al, 1979; Stubbs et al, 1978) and beta-endorphins (Kato et al, 1978; Guillemin, 1978) have been shown to stimulate prolactin, growth hormone, thyrotrophin but depress luteinizing hormone, thyroid stimulating hormone and FSH secretions. It has also been demonstrated that beta-endorphin releases oxytocin (Haldar et al, 1979) and D-ala (2)-enkephalinamide releases vasopressin (Bisset et al, 1978). Beta-endorphin stimulates corticosterone synthesis in isolated rat adrenal cells by binding to the adrenocorticotropic hormone receptors (Shanker and Sharma, 1979). ACTH and beta-endorphin are stored in the same secretory granules of anterior pituitary (Weber et al, 1978) and are secreted concomitantly (Guillemin et al, 1977). There is a feedback loop between the beta-endorphin (or ACTH) and corticosterone (or cortisol); as dehydration (Mata et al, 1977; Cox et al, 1978) or dexamethasone administration (French et al, 1978) reduces beta-endorphin and ACTH secretion, while adrenalectomy or metyrapone administration elevates plasma beta-endorphin levels by 7 fold (Hollt et al, 1978; Tseng and Li, 1979). Endorphins in the pituitary seem to be releasing factors or precursors to releasing factors of corticosterone. Furthermore, in normal animals, naloxone alone produced opposite effects in modulating the hormonal releases (Meites et al, 1979). This suggests that endorphins participate in regulating normal secretions of the pituitary. Enkephalinergic cells are found in the islets of Langerhans and beta-endorphin and enkephalins have been shown to inhibit pancreatic secretions and increase insulin and glucagon release (Meites et al; Konturek et al, 1978). A large number of opiate receptors are found in the intestine and stimulation of enkephalinergic neurons in the small intestine suppresses intestinal motility. Beta-endorphin produces hypothermia and alpha-endorphin produces hyperthermia (Guillemin et al, 1977). Finally met-enkephalin has been shown to depress heart rate, blood pressure and respiration when applied to brain stem (Laubie et al, 1977). All the above results indicate that endorphins are involved in the physiological regulation of the cardiovascular system, the respiratory system, the digestive system and body temperature.

c) Mental illness

Endorphins have been shown to produce profound physiological effects when administered directly into the brain. They cause "wet-dog" shakes, sedation, catatonia and catalepsy (Guillemin et al, 1977; Frenk et al, 1978; Elazar et al, 1979). Seizures and long lasting epileptiform changes in EEG are observed with injections of met-enkephalin (170 ug/injection) into or near the forebrain dorsomedial nucleus of the thalamus (Frenk et al, 1978) or leuenkephalin (1-20 ug) into the dorsal hippocampus (Elazar et al, 1979). Low (non-analgesic) doses of beta-endorphin induces non-convulsive limbic seizures (Henrikson et al, 1978) and catalepsy (Moroni et al, 1978).

Beta-endorphin has been used to induce akinesia in animals and a significant increase in concentration of 5-hydroxytryptamine is observed in the midbrain (Izumi et al, 1977); the induced akinesia can be reversed by methylmorphine, L-dopa and partially by L-

amphetamines. It has been demonstrated that beta-endorphin or δ -ala (2)-enkephalin analogue increase serotonin metabolism in hypothalamus and dopamine turnover in neostriatum, tuberculum olfactorium and nucleus accumbens (Fuxe et al, 1977; Van Loon et al, 1978; Biggio et al, 1978). It is proposed that there is a feedback relationship between endorphin neurons and dopaminergic neurons.

The potent and divergent behavioral responses of animals to naturally occurring substances (especially beta-endorphin which has a more marked, prolonged effect even at very minute doses) indicate that alteration in their homeostatic levels might have etiological significance in mental illness. CSF endorphin levels were found to be higher than normal in depressed or schizophrenic patients (Terenius et al, 1976) and lower than normal in chronic pain patients (Terenius, 1977). This might explain how electric shock therapy has therapeutic effect on schizophrenic patients as it might reduce their endorphin levels. Chronic schzophrenic patients under neuroleptic medication show rapid inactivation of C.S.F. endorphin (Dupont et al, 1978). Type II opiate receptors (or K and δ receptors) may be the ones that mediate sedation, dysphoric and hallucinatory syndromes (Pert et al, 1979 and Martin et al, 1977). High doses of naloxone, have been reported to eliminate hallucinations in schizophrenic patients (Gunne et al, 1977; Watson et al, 1978). Leu (5)-B-endorphin are only detected in kidney dialysis of schizophrenic patients and it has been suggested that this may play a role in the chemical etiology of schizophrenia (Govoni et al, 1979).

Recently, Van Ree and colleagues (1978) reported that des-Tyr (1)-gamma-endorphin is a neuroleptic substance; it contains no opioid activity but can interact with neuroleptic binding sites in various areas of rat brain. Beta-endorphin and other opiates have no affinity for neuroleptic binding sites (Czhonkowski et al, 1978). Des-Tyr (1)-gamma-endorphin is found to be very effective in treating schizophrenic patients (Verhoeven et al, 1979; Van Ree et al, 1979). They postulate a dual system that endorphin reduces stress and des-tyr-gamma-endorphin produces sedation.

d) Behaviour

Endorphinergic neurons are shown to have extensive interactions with acetylcholine, dopamine, 5HT, noradrenaline and GABA neuronal systems in the brain (Costa et al, 1978). Beta-endorphin increases the turnover rate of GABA in the substantia nigra and lobus but decreases the turnover rate in nucleus caudatus (Moroni et al, 1978). The turnover rate of acetylcholine is decreased by beta-endorphin in cortex, hippocampus, nucleus accumbens and globus pallidus but not in nucleus caudatus (Moroni et al, 1977). Met-enkephalin and leu-enkephalin also inhibit the spontaneous release of acetylcholine from the cerebral cortex in vivo (Thamandas et al, 1977). This indicates that endorphins may act as presynaptic inhibitors of cholinergic neurons. The interactions of endorphins with other neuro-substances may preserve a homeostatic state of bodily functioning which helps to maintain normal behaviour. Evidence has shown that alpha-endorphin induces tranquilization, gamma-endorphin produces agitation and sometimes violent behaviour, beta-endorphin causes catatonia (Costa et al, 1978). and hypotension which is reduced by PCPA (Lemaire et al, 1978), and des-Tyr (1)-gamma-endorphin improves schizophrenia (Van Ree et al, 1979). All these effects suggest that the endorphinergic systems are probably involved in maintaining normal behaviour and that a malfunction of this system may result in psychiatric illnesses or abnormal behaviour. For example, evidence indicates that overeating is probably caused by the abnormally high pituitary beta-endorphin level (5 times higher than normal animals) in obese mice (ob/ob) and rats (fa/fa) (Margules et al, 1978). Naloxone selectively abolishes overeating in these genetically obese mice and rats (Margules et al, 1978).

Endorphins, like morphine, produce euphoria. It has been found that leu-enkephalin induces higher rates of self-administration in the self-administered bar-pressing behaviour as compared to met-enkephalin when they are injected into the limbic areas in rats (Belluzzi et al, 1977). It is possible that leu-enkephalin is a major intrinsic substance that elicits pleasure and reward-system.

Social and sexual behaviours are also affected by endorphins. Beta-endorphin increases grooming activity, decreases mounting and increases the length of time before ejaculation in rats (Meyerson and Berg, 1977). A high dose (6 ug) of D-Ala (2)-met-enkephalinamide (analogue) also suppresses the copulatory behaviour of rats but a low dose (3 ug) increases mounting and intromission latencies (Quarantotti et al, 1978).

e) Addiction

Endorphins, like morphine, produce tolerance, cross-tolerance and withdrawal effects on long-term treatment, i.e. they are also addictive (Tseng et al, 1976). Endorphin can modify the relative activities of adenylate cyclase and guanylate cyclase which are two enzymes floating freely in the double layer of lipid molecules that make up the cell membrane. These two enzymes will be activated to synthesize cyclic AMP and cyclic GMP when appropriate neurotransmitters bind to the membrane receptors. Often, these two enzymes display antagonistic action in mediating the same hormonal effect inside the cell. In neuroblastoma-glioma hybrid cell cultures, long-term exposure to opiates or endorphins will induce an abnormally large production of adenylate cyclase which enhances cyclic AMP synthesis. As a result, more opiates or endorphins are

required to produce decreases in cyclic AMP levels (Klee et al, 1975). On withdrawal of opiate or endorphin, cyclic AMP levels increase markedly owing to the abnormally high quantity of adenylate cyclase. This increase in cyclic AMP level correlates biochemically to the withdrawal symptoms. The increase in the cyclases during and after long-term opiate or endorphin treatment may act as a biofeedback mechanism which will change the firing rate of endorphin neurons, inducing the development of tolerance and physical dependence. It has been shown that SPA of PAG or chronic beta-endorphin administration cause tolerance which can be reversed by 5-hydroxy-tryptophan (Hosobuchi et al, 1977; Hosobuchi et al, 1978). Thus serotonin may play an important role in addiction and analgesia.

Conclusion

These endogenous ligards, called endorphins (endogenous morphine) are peptides which have a similar effect to morphine in inducing analgesia, euphoria, relieving depression and affecting other important metabolic functions. Brain endorphin levels are independent of pituitary endorphin levels. Biochemical and anatomical evidence indicates the existence of two separate opioid systems in brain: beta-endorphin containing neurons and enkephalin containing neurons which constitute two separate groups of brain cells. □

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Ankle Sprains:

An approach to identification and treatment

Brian Reiss 8T2

Ligamentous injuries about the ankle are a very common problem and considerable controversy exists regarding the best way to treat them. An approach to this problem would be to first decide whether or not the injury is significant, and then to choose one of three basic modes of treatment that are available: simple strapping, plaster immobilization and operative repair.

Various viewpoints with their supporting evidence from the literature and a retrospective study will be presented here. It will be proposed that in the light of a suggestive clinical picture, the presence of a difference of talar tilt between injured and non-injured ankles is indicative of significant damage to the lateral ligaments. Also to be put forward is the suggestion that the ankle with significant tilt should be casted and the one without should be strapped.

Definition of terms

Ankle Sprain in this article means soft tissue injury around the ankle with possible rupture of the ligaments.

Chronic Sprain (weak ankle) implies to a variable degree, some combination of persistent swelling and tenderness following injury, recurrent sprains, and complaints of weakness and instability.

Talar tilt angle (tilt)[11] means "the angle formed by the opposing articular surfaces of the tibia and talus when these surfaces are separated laterally by a supination force..."[28] (Others measure the absolute separation in millimeters).

A Varus Stress Radiograph is taken in an anterior to posterior direction, perpendicular to the bimalleolar axis, with an inversion force applied to the foot[18] or calcaneus [28, 29].

The Anterior Drawer Sign (of the ankle) is the appearance of the talus subluxing forward beneath the tibia when a stress is applied across the ankle in a sagittal plane.

An *Anterior Stress Radiograph* is a lateral view of the talus in the mortise obtained while the patient's heel is fixed in position and a posteriorly directed force is applied to the distal end of the tibia.

Arthrography is taken here to mean radiographic examination of a joint with the aid of intra-articular contrast media.

A Peroneal Tenogram is a radiograph taken after injection of contrast medium directly into the peroneal sheath.

A "Stress Tenogram" is an investigation that combines stress inversion and anterior stress radiographs with the peroneal tenogram.

Normal anatomy of the ligaments and capusle of the ankle joint

The capsule[5] is attached at the bone cartilage border of the tibia, fibula and talus. It is lax anteriorly and posteriorly and therefore allows flexion movements to occur. Collateral ligaments reinforce the capsule medially and laterally.

There are three distinct ligaments on the lateral side of the ankle:

1. the Anterior Talofibular Ligament (ATFL);
2. the Calcaneofibular Ligament (CFL);
3. the Posterior Talofibular Ligament (PTFL);

In addition to the above named ligaments there are others not yet mentioned. One of interest is the talocalcaneal ligament because it will be mentioned again later with regard to the surgical repair of the lateral ligaments of the ankle.

Clinical Material

All of the people in this study were University of Toronto students (except one) seen in the Health Service Athletic Clinic by an orthopedic surgeon or by a trainee under supervision. Records from the academic sessions between the fall of 1978 and the spring of 1980 were examined in order to locate people who sustained an ankle sprain for the first time and whose clinical work up included varus stress radiographs. Of the thirty-six records obtained, sixteen people could not be contacted, and of the twenty people asked to come in for follow up, six consistently failed to keep their appointments. The remaining fourteen were divided into two groups on the basis of talar tilt as seen on stress radiographs taken around the time

of the injury. Five of these were considered to have a significant[21] difference of tilt between the injured and uninjured ankles and nine were not.

The first of the two groups consisted of three males and two females with an average age of twenty-four (range 19 to 36 years). All had a difference of at least ten degrees of tilt between ankles (average of 17.6 degrees with a range of 10 to 27 degrees) and an absolute tilt in the uninjured ankle of at most ten degrees (one with 10 degrees, the balance with 2-3 degrees). Three people in this group were seen within 24 hours and put in a below the knee walking cast with the ankle in a valgus position[21]. All were seen and casted within 48 hours, keeping the cast on for four to five weeks.

The second group consisted of eight males and one female with an average age of twenty (range 18 to 23 years). All lacked significant talar tilt. All were seen within four days (average of two days) of injury and strapped[21], keeping the tape on for two to three weeks.

Findings on Follow-up

Group 1 (tilt positive at time of injury)

On average twenty months elapsed between the time of injury and follow-up (range of 15 to 26 months). All of the patients considered the function of their ankle to be satisfactory and none curtailed their activities due to symptoms.

Three out of five had some degree of functional instability. (one of these went over on the uninjured ankle more than the injured and one had mild trouble on rough ground. Only one felt he had to tape his ankle for sports, and in fact was the only patient in this group to have reinjured his ankle between treatment and follow-up).

None had continued pain in their ankle but two would sometimes get a mild ache in their ankle after exercise and two others would get a slight degree of swelling, all of which would be gone by the next morning.

All were able to return to the sport of their choice. Some decreased the level of their activity but cited leaving school rather than their ankles as the cause.

Objectively, none had a clinically noticeable degree of difference in talar tilt between ankles, and all had insignificant tilts radiographically (less than three degrees). Only one person seemed to have an increased range of motion in the subtalar joint of the injured ankle as compared to the uninjured but that was also the person who had reinjured his ankle (then treated by strapping out of clinic).

Group II (tilt negative at time of injury)

An average of twenty-three months elapsed between the time of injury and follow-up (range of 17 to 30 months).

Five out of nine had some degree of functional instability. (Two of these experience this in both ankles. Two others wear high backed shoes, and one other tapes his ankle for sports).

None had continued pain in their ankle. (Two considered their ankle to be tender over the lateral margin. Three would get some aching in their ankle after exercise which would be gone by the next morning).

Only one patient would get some transient swelling of his ankle after exercise. (this person also got an aching after exercise, was tender on the lateral aspect and taped his ankle for sports and has reinjured it a couple of times).

All continued with the sport of their choice. Some decreased their level of activity but again cited leaving school rather than their ankles as the cause.

Objectively, only one had a slightly greater clinically detectable tilt of the injured versus the uninjured ankle. One other had a mildly decreased range of inversion on the injured as compared to the uninjured side.

Clinical Presentation

About 35 to 40 percent of patients will report having sustained one or more previous sprains in the past [4, 7, 30]. Most ankle sprains occur during some form of athletic or work related activity [7, 22] and are usually the result of a forced inversion[12] though hyperplantarflexion and internal rotation[7, 12] are sometimes mentioned. Initially there is a transient but substantial pain[7] which intensifies in 12 to 24 hours[11].

Immediately following the injury there are few physical findings[11]. After a period of time a variable amount of swelling and ecchymosis can be seen and tenderness, both direct and indirect, can be elicited. These findings, in addition to greatly augmenting the significance of various clinical maneuvers to be considered later, are to some degree, useful in localizing and delimiting the extent of damage.

Swelling within a few hours of the sprain [7, 11] (some say within the first half hour[20])-intra and extra articular bleeding in combination with an inflammatory reaction lead to swelling[7] particularly prominent over the lateral malleolus[11] though it is often diffused over the ankle joint and midfoot[7]. It is always found at the site of ruptured ligaments[7] and in such cases is usually greater than two centimeters by three centimeters in area[29].

Ecchymosis is found in about 50 percent of patients[7] and often does not become visible until 12 to 24 hours after the injury[7, 11]. Initially the discolouration is confined to the vicinity of the damaged ligament but over a period of a couple days it transforms into a blotchy purplish patch found along the margin of the foot or above the toes[7].

Tenderness is the most informative guide to the site of injury[7, 29] and is invariably found over a ruptured ligament[7]. In a substantial proportion of cases though the tenderness extends to adjoining ligaments[7]. To overcome this, the point of maximum direct tenderness has been used to identify which ligaments have been torn and has been found to correlate well with surgical findings[29]. Indirect tenderness, or pain localized

to the lateral aspect of the ankle, can be elicited by internal rotation or inversion when the ATFL is torn, and by adduction when the CFL is torn[7]. Sometimes though all movements of the foot are painful making interpretation difficult.

Approximately 75 percent of the people with this sort of clinical presentation will have some form of lateral ligament rupture[5]. Of these, an isolated complete tear of the ATFL is most commonly found [1, 26] (69 percent[5]) followed by a complete rupture of the CFL in addition to the ATFL (16 percent[5]) with other combinations making up the difference. Some feel that the ATFL is always the first to tear and that the CFL is never torn in isolation[5, 18, 30]. Others say this is true except when the ankle is in neutral, at which time tearing of the CFL may occur first[12, 25].

The PTFL was found to be intact in all but the rarest of cases[2, 3, 4, 5 11, 12, 29]. The deltoid ligament will not be considered here.

In the past, some have thought that complete ligamentous rupture was always associated with avulsion of a fragment of bone. In a more recent series, however, avulsion was found in only fifteen of the 96 cases that had complete tears of one or more ligaments[5]. Therefore, the absence of avulsed fragments on plain radiograms cannot be regarded as a reliable indication of non-occurrence of complete ligamentous rupture[5].

Treatment

Three main lines exist for treatment of acute ankle sprains.

Strapping is the application of adhesive tape[21] (elastic bandage[8]) in figure of eight turns around the ankle. When the swelling subsides or after about two weeks[21] the strapping can be removed. Strapping has little stabilizing function[8].

Casting means the application of a below knee walking plaster with the foot in neutral dorsiflexion and some eversion [8, 21]. The cast is usually applied at the first visit but if there is severe swelling is can be delayed a couple of days[8]. It can be removed anytime after three[8] to six[21, 29] or more weeks later.

Surgical repair, in the case of all the studies of acute sprains described below, implies direct reapproximation of ligaments with fine continuous sutures. If an avulsion fragment is present, then it is replaced by suture through drill holes. After surgery a below knee walking cast is applied as described above[8].

These three modes have been compared in clinical studies by many investigators using almost as many sets of criteria for determining the state of the sprained ankle both before and after treatment.

There are also several studies that look into the predictive: value of the criteria in question, but even among these there is a lack of unanimity. The varus and anterior stress tests are two that have received much attention in the past and will be discussed presently.

Physiologic variation in the degree of talar tilting after the application of a varus stress definitely exists[25, 28] and can be demonstrated reproducibly without excessive force[25], but there are limits to the extent of this variability. In addition, by considering the degree of tilt in the light of other available data, its significance can be greatly augmented.

The next step that can be taken is to measure the difference in tilt between the injured and uninjured ankles. Average talar tilt in right and left normal ankles has been found to be the same but in any one individual it need not be identical[25]. However, the physiologic asymmetry was found to never exceed fifteen degrees[25] (nineteen degrees[28]) and was usually less than ten degrees[25].

Another observation concerning the difference between injured and uninjured ankles is that in the case of the uninjured ankle, the leg and talus, as a unit, externally rotate relative to the calcaneus when an inversion stress is applied. No rotation occurs if the CFL is damaged. This rotation can be seen radiographically as a superimposition of the lateral malleolus on the tibia and talus[25].

Some feel that the rotation is due to the configuration of the talocalcaneal joint[28] and others believe it is due to the orientation of the CFL[25]. It is likely that both are contributory and in either case rupture of the CFL probably allows disruption of the normal configuration of the subtalar joint under varus stress[25, 26].

Finally, if all of the above is considered in the light of a highly suggestive clinical presentation, then a variation in talar tilt between ankles is most likely significant[11], especially if there is a large incongruity. Occasionally tilt may be detected clinically (i.e. without radiographic examination) as a palpable gap between the lateral malleolus and the talus when the ankle is inverted[21].

Significance of Talar tilt

Surgical findings indicate that if a significant tilt exists (a difference of three or more degrees between ankles[11]), then a tear of at least ATFL[11], both ATFL and CFL[2 3 29] or some combination[15] always exists. In other words a significant tilting of the talus necessarily implies a ligamentous tear. In addition, the degree of tilt increases as the severity of ligamentous damage worsens[13, 14, 27]. One investigator obtained a good correlation[27] but others found much overlap in ranges of tilt for each degree of injury[13, 14]. The reverse, that is the lack of tilt implying the absence of ligamentous damage, cannot necessarily be concluded. In one study of which all the ankles had some ligamentous disruption, very few were demonstrated to have a significant tilt[7].

Some feel that in the absence of tilt, an anterior stress radiograph should be examined to decide on the state of stability of the ankle[18]. But, relative to the talar tilt, the anterior drawer sign and anterior stress radiograph have had little investigation. Even so, an association between anterior subluxation and rupture of the ATFL has been noted both at surgery and by arthrographic examination[2, 5, 7, 18, 30].

In acute sprains, the anterior drawer sign could not be elicited in many ankles without anesthesia but with anesthesia all the ankles with an ATFL tear had demonstrable forward subluxation[7, 30]. In chronic sprains, all of which had surgical evidence of an old tear of ATFL, only 73 percent (44/60) had an obtainable anterior drawer sign.[9]. The failure of some of these ankles to have a drawer sign was postulated, in part, to be due to muscular tension and a thickened and fibrosed perisynovial tissue which stabilized the ankle preventing forward dislocation[9]. The same investigator found very few anterior drawer signs in normal ankles.

Because of its configuration, the ankle is a highly stable joint whose stability is increased in the weight bearing position[8]. In the dorsiflexed foot the malleoli are in firm contact with the talus and resist talar tilt[29]. Other limiting factors in detecting tilt and anterior subluxation are; surrounding tissues that limit opening; apprehension and voluntary resistance; and of course skill of the physician and timing of the radiograph with respect to application of the stress[29].

Assuming these factors can be overcome, another line of thinking comes to mind. Since these maneuvers test the stability of the ankle and since different degrees of injury may produce differing amount of instability in different patients[19] then instead of just correlating the results of these tests with the pathologic state of the ankle ligaments, it would be logical to look at the overall predictive value of these tests with regard to the final outcome after treatment.

Unfortunately, little has been done to investigate the relationship between the presence or absence of pretreatment anterior subluxation and results of various treatment entities. There is though, a fair amount of information available to allow some correlation between the existence of pretreatment talar tilt and the final outcome of treatment.

Some investigators believe that anesthesia is needed before a reliable varus stress test can be performed [10, 30] but workers in Toronto have found it unnecessary to use either local or general anesthesia. The procedure is carried out slowly and gently after giving a thorough explanation to the patient[21]. If more than twenty-four hours have passed since the injury occured, other methods such as arthrography, may have to be considered to evaluate the status of the ankle[21].

Results

The results of studies that utilized ankles having a *significant talar tilt* will now be considered. In most of these studies the residual symptoms were found to be quantitatively comparable between surgically and cast-treated groups with some giving the surgical group a slight advantage. (But the surgical group also has some unique complications and symptoms to be discussed later).

In almost all cases a satisfactory result was obtained by casting. For those few that end up with severe symptoms after initial cast treatment, surgical repair can be offered with the knowledge that good results can still be obtained[9, 23].

One study compared strapping, casting and surgery[15]. The strapped group had a higher percentage of significant persistent tilt but none of the groups contained grossly unstable ankles. In all three groups approximately forty percent of the patients had functional instability. Only twenty-five percent of the patients in the surgical group were symptom free, whereas about fifty-five percent of the patients in the other groups were asymptomatic. The validity of conclusions drawn from this data must be suspect since the average degree of tilt at the time of injury varied considerably between the treatment groups, being greatest in the surgical and least in the strapped group. (In addition, the percentage of patients with residual symptoms was considerably higher than that obtained by other workers).

Next to be considered are the results of studied that utilized ankles *without a significant talar tilt*. One major study[8] contained a mixed population, most of the ankles being tilt negative but not all.

Surgical results obtained in this investigation were considerably better than results obtained using the other treatment methods[8]. Three percent in the surgical group had mild symptoms (though more had symptoms as a result of treatment which will be discussed later), whereas approximately twenty percent had symptoms in the casted and strapped groups with no significant difference between these latter two.

Others have obtained somewhat poorer results after strapping. In one of these studies the patients were only followed for at most five months[26] and in the other, elastic strapping was used and 42 percent had previous sprains.

Complications

The three treatment regimes, for both tilt positive and negative ankles, can also be compared with respect to complications produced. Besides time lost from "work", which was least with strapping and greatest with surgery[8, 15] (strapping eighteen, casting thirty-four and surgery forty-five days[8]) strapping and casting have virtually no complications. The following though, are possible complications of surgical repair with the approximate incidence (when known), indicated in parentheses:

- limitation of range of motion (25 percent[15], 5 percent[8])
- superficial necrosis of skin in area of incision (10 percent[30])
- hypoesthesia distal to scar (ten percent[8, 30])
- dysesthesia in area of scar[8]
- operative infection and osteoarthritis[8].

All the treatments have approximately the same poor rate of success in alleviating functional instability which is also the most common residual symptom[31]. Such factors as, for example, calf muscle weakness, residual talar tilt or an-

terior instability, tenderness over the lateral ligaments or a decreased range of motion have been shown, in some cases, to make functional instability worse but none have been demonstrated to be the cause[3, 16]. Likewise, mechanical stability does not afford any guarantee against its occurence[31].

If one considers that many articular nerve fibers terminate in mechanoreceptors in the capsule and ligaments of joints, then after ligamentous and capsular trauma it is comprehensible that partial joint deafferentiation may occur and may be permanent regardless of the treatment used[17]. Stabilization would then be impaired and the foot would tend to "give way"[17]. Some agree with this theory[3, 31] but others have been unable to detect a proprioceptive defect in their series of patients[30].

For the present, in the case of tilt-positive ankles, casting is a valid first line treatment and even though no study conclusively proves its superiority over strapping, it is recommended because the weight of evidence tends toward the former.

For tilt-negative ankles, strapping provides satisfactory results which are comparable to casting and for now, should be the recommended treatment mode.

If after conservative treatment the condition of the patient's ankle is not satisfactory, the direct suturing of the torn ligaments, with or without supplementation by a flap from the talocalcaneal ligament, can be performed yielding good results[9, 24]. A large variety of other repairs using tenodesis, free fascia or free tendon grafts have been used in the past but have many drawbacks[2, 9, 10, 24].

The question of whether any harm is done by not treating the acute injury surgically can now be addressed. Even though on average casting provides results almost equal to those of direct surgical repair, in the hands of some[8], suturing can be superior. In addition, it has been shown that a proportion of chronic sprains go on to develop problems after ten or more years duration[29].

In answer, surgical repair, especially of the ATFL, can reverse these changes in most cases[9, 23], and if instituted before the degeneration of ankle function has occured it can be prevented in the majority[9]. Also, by attempting conservative treatment first, unnecessary surgery (which would swamp the available surgical units[8]) and its complications can be avoided.

(Continues on page 61)

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Ontario Medical Association

Degenerative Hip Disease

Lyall Gorenstein 8T3
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"A mind that understands principles will work out the Methods."
Hugh Owen Thomas

Degenerative joint disease is an extremely common, non-inflammatory, progressive disorder of joints. Degenerative hip disease (D.H.D.) is the most disabling and one of the commonest forms of degenerative joint disease. At least 175,000 Americans aged 65 years and over are incapacitated by D.H.D.[14] The patient with D.H.D. may be plagued with constant pain, deformity, restricted motion and most significantly loss of independence. This type of physical disability accounts for a significant number of days "off work."

The management of D.H.D. is based on knowledge of normal hip biomechanics. The non-surgical and surgical methods in treating D.H.D. are aimed at restoring pain-free hip function. Since, a knowledge of hip biomechanics is so essential in understanding the pathogenesis of D.H.D. and its management, the major emphasis of this article is to provide a biomechanical perspective of D.H.D.

Biomechanics of The Normal Hip

During the monopedal support phase of the walking cycle, one limb supports the total body weight. At this instance the force on the hip joint is maximized. In the other phases of the cycle, the force on the joint is less. The force acting on the femoral head (force R) passes through its centre of rotation (CR), perpendicular to the articular surface (figure 1)[2, 13]. Force R is the vectorial sum of forces M and K. Its direction is 16 degrees in the forntal plane, and varies in the sagittal plane throughout the walking cycle, from antero-inferior, to postero-inferior. The magnitude of force R is almost four times the body weight in the monopedal support phase.

Force K is equal to the body weight minus the weight of the supporting limb. It acts medially to the hip joint through the body's centre of gravity (CG). Its magnitude depends on the individuals weight.

Force M is produced by the abductors contracting to maintain the pelvis horizontal while the opposite limb is elevated. The magnitude of force M depends on the relationship of the lever arms through which forces M and K act, b1 and b2 respectively.

The body weight lever arm, b2, is normally three times longer than the abductor lever arm b1 . In order to maintain the pelvis horizontal in the monopedal support phase, force M must be three times greater than force K. Expressing this algebraically, (Mb1 + Kb2 - 0). In other words, the sum of the moments about CR is zero[2, 13].

Pauwels describes a counter-resultant force, R', acting in the opposite direction to R at all times. Force R' is resolved into two components, P and Q. Force P is direc-

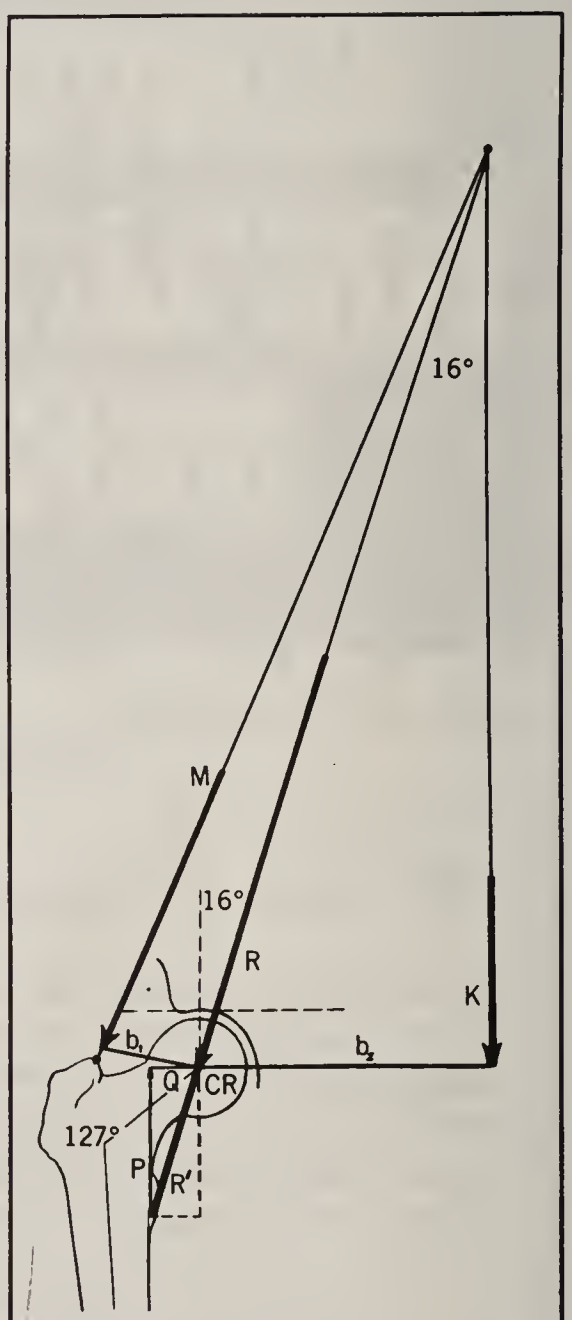

Figure 1: Forces and Biomechanics about the hip joint in the monopedal support phase of gait.

ted vertically, always perpendicular to the weight bearing surface, and is compressive. Force Q acts parallel to the weight bearing surface, directed medially, pushing the femoral head into the acetabulum (figure 2).

The femoral head and acetabulum are physiologically stressed by pure compression because force R and its counter force R' act perpendicularly to their surfaces. The compressive stresses are uniformly distributed through the joint because both surfaces are covered with articular cartilage. The magnitude of the compressive stresses depends on 1) the magnitude of force R and 2) the joint surface area transmitting the force. The latter factor has a greater influence on the magnitude of compressive stresses and the pathogenisis of D.H.D. [13].

Equal distribution of compressive stresses in the hip can be seen radiographically. Figure 2a, is a schematic representation, showing equal density of subchondral bone in the acetabular roof. If the stress to one area of the joint is increased, it produces greater density of the subchondral bone according to Wolff's law (figure 2b).

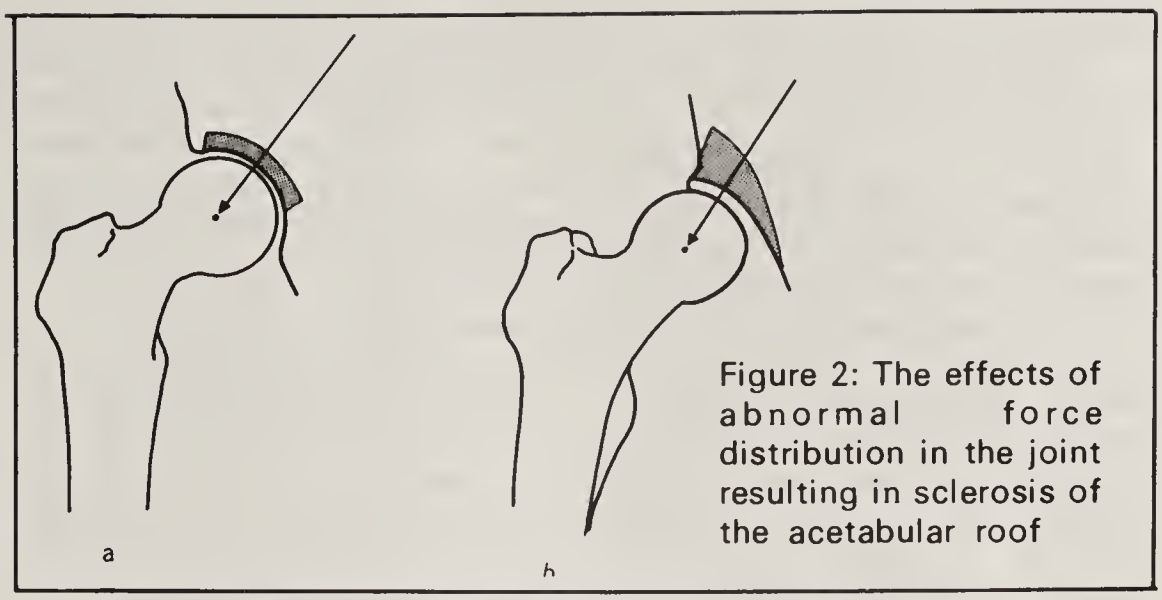

Figure 2: The effects of abnormal force distribution in the joint resulting in sclerosis of the acetabular roof

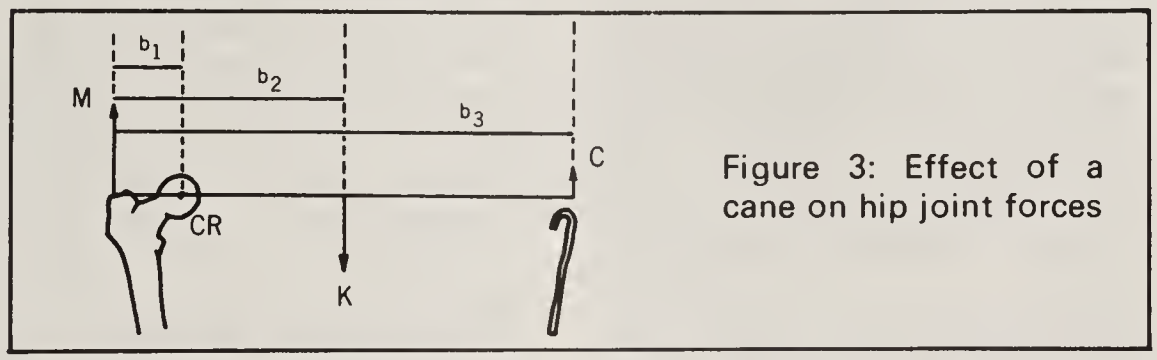

Figure 3: Effect of a cane on hip joint forces

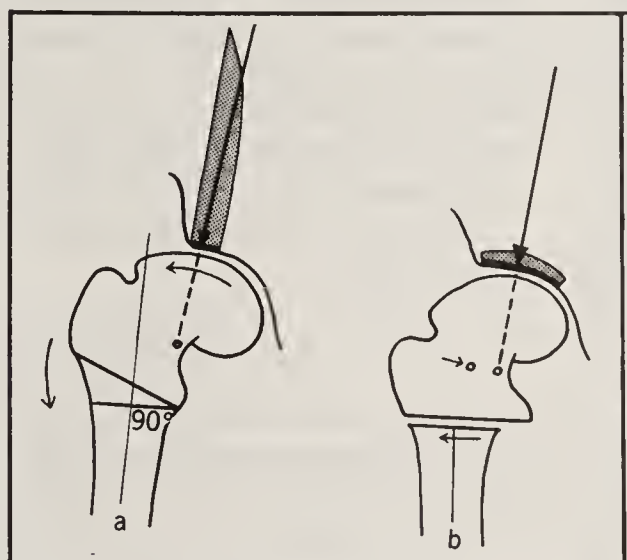

Figure 4: the effect of a Femoral varus osteotomy on abnormal hip forces secondary to a valgus deformity.

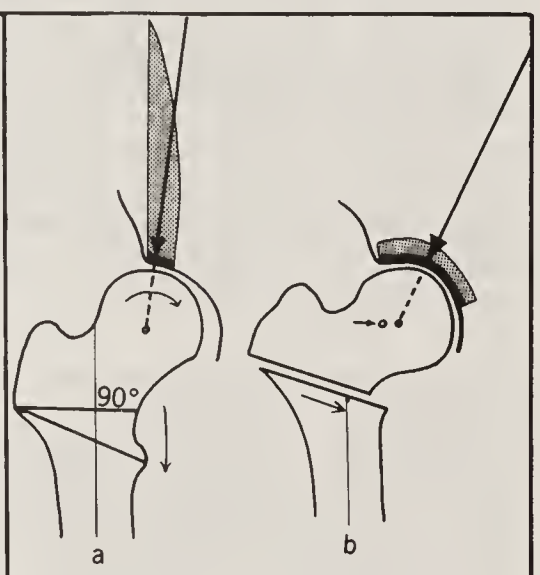

Figure 5: The effect of a Femoral valgus osteotomy on abnormal hip forces.

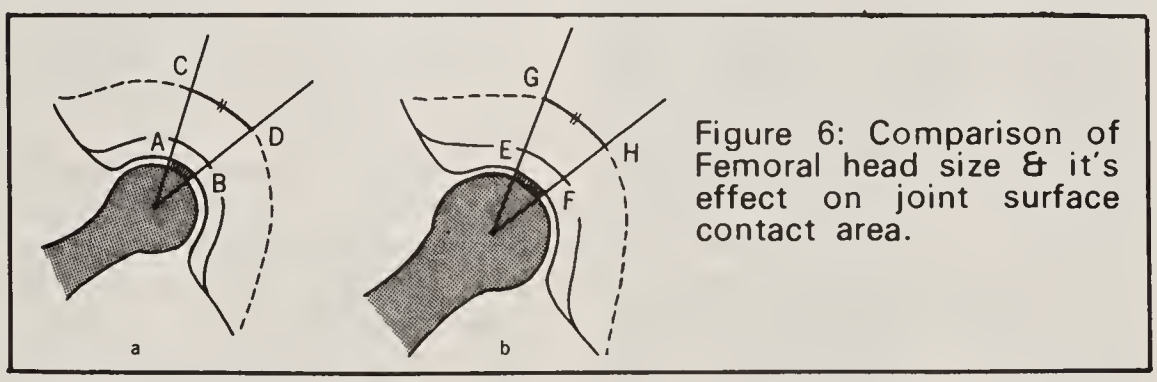

Figure 6: Comparison of Femoral head size & it's effect on joint surface contact area.

Pathogenesis

Bone and cartilage depend on organic substrates and normal mechanical stresses for their maintenance. Stresses on articular cartilage must remain within certain limits. If the upper limit is surpassed, it degenerates exposing subchondral bone. When stress is reduced below the maintenance requirements, cartilage is replaced by bone, and ankylosis ensues[15]. Pauwels claims that bone formation and resorption depend on the mechanical demands placed on it. Reducing function (immobilization) decreases osteoblastic activity resulting in osteoporosis. Increased function stimulates osteoblasts producing osteosclerosis. Chronic overuse produces microfractures that stimulate osteoclast activity resulting in subchondral pseudocyst formation.[2]

A physiologic equilibrium exists between the magnitude of articular pressure and the resistance of bone and cartilage to mechanical forces. The equilibruim can be upset by either decreasing tissue resistance or by increasing articular stress. D.H.D. results from disruption of this equilibrium, and in most cases both mechanisms interact to disrupt the equilibrium and ac-

celerate the disease process[13].

A) Decreasing Bone and Cartilage Resistance

Many disease processes reduce the ability of cartilage and bone to respond to normal articular stress; the most common being idiopathic D.H.D. which accounts for approximately 50%. There are some authors who believe that there is no such entity as idiopathic D.H.D., but that it may represents an etiology not yet been accounted for; for example, a mild undetected infection. There are biochemical changes in the articular cartilage ground substance resulting in its degeneration and the exposure of collagen fibrils. Simultaneously chondrocyte lysis occurs followed by resorption and microcyst formation. There is an attempt at cartilaginous regeneration, but it cannot keep pace with the destructive process. Eventually, the cartilage erodes away, exposing subchondral bone. With asymmetrical loss of cartilage from areas of the articular surface, stress is not uniformly distributed. In areas of increased stress, tissue resistance is surpassed and degeneration occurs.[13] [16]

(B) Increasing Articular Stress

Increasing articular stress beyond tissue resistance results in D.H.D. The magnitude of the compressive stress can be increased by 1) increasing the magnitude of force R or 2) Decreasing joint surface area. This occurs secondary to joint disruption by a disease process, such as congenital dislocated hip. The resulting joint is incongruous, its surface area reduced, and its biomechanics disrupted. This eventually leads to the pathologic changes typical of D.H.D.

The histopathology in D.H.D., is the same regardless of the mechanism upsetting the physiologic equilibrium. In the zone of overloading (increased stress) there are signs of remodelling. Initially bony sclerosis dominates, but as articular stress becomes excessive, subchondral pseudocyst formation occurs. Surrounding the zone of overloading is an area of reduced stress, where there is excessive bone formation activated by the remodelling process in the zone of overloading[13].

Classification of D.H.D.[5, 15]

(A) Congruous: decrease in biological resistance of tissue

i) Idiopathic: a) monoarticular b) polyarticular

ii) Infection: eg. septic (staph. aureus 90%), T.B.

iii) Rheumatoid Arthritis

iv) Miscellaneous: eg. hemophilia, ochronosis.

(B) Incongruous: excessive joint pressure

(1) *Intra-articular:* irregularities of joint surfaces.

i) Intra-articular fractures, cartilage lesions.

ii) Osteonecrosis of bone

a) idiopathic: eg. Legg Perthes.

b) acquired: eg. steroids, fat embolism, micro-fracture.

iii) Loose bodies

iv) Slipped femoral epiphysis

v) Dysplasia:

a) congential: eg. congenital dislocated hip.

b) acquired: eg. secondary to polio.

(2) Extra-articular: excessive joint loads and misalignment of joint forces.

i) Bony deformity:

a) malunited fracture eg. femur, tibia

b) systemic bone disease eg. osteomalacia, Paget's.

ii) Leg length discrepancy - D.H.D. on side of longer leg.

iii) Loss of protective feedback:

a) congenital indifference to pain

b) tabes dorsalis, diabetes, syringomyelia

iv) Remote causes:

a) obesity

b) immobilization

c) occupational over-use

Clinical Presentation

- Pain - the cardinal symptom
- Difficulty using stairs, putting shoes and socks on
- Limited walking distance
- Limp - Trendelenburg gait
- Trendelenburg sign
- Walking aid
- Muscle contractures
- Decreased range of motion

Pain: Most patients complain initially of aching in the hip region, which may be referred to the groin, the lateral aspect of the thigh and frequently as far distally as the medial aspect of the knee. Pain is triggered by weight bearing especially following a period of joint rest, thus pain may begin either in the morning or after sitting. With increasing activity the symptoms become less severe and are relieved by rest. Later in the course of the disease pain may be severely aggravated by activity and unrelieved by rest. Eventually, the patient becomes severely handicapped and experiences constant pain.

Some patients will describe stiffness or fatique initially. Also, it is important to realize that 20% of patients with D.H.D. will present with pain in the distal thigh or knee region[17].

The diffusion of pain around the hip stems from the fact that the obturator and sciatic nerve fibers which supply the hip joint also innervate skin, muscle and bone. Pain in the hip sensitizes the obturator nerve which initiates a pain reflex resulting in the contraction of muscles. This is mainly demonstrated by adductor muscle contraction, whereby over a period of time fibrous tissue supervenes and contractures are produced. Since the adductors cause adduction, external rotation and flexion, contractures are produced in these positions. The adduction contracture leads to a relative shortening of the limb, but absolute loss of length follows cartilage loss.

Although joint mobility may become extremely limited, spontaneous bony ankylosis does not occur and pain persists. The progression of the disease in quite variable. In some patients the pain may become incapacitating in less than one year, whereas others may have little increase in hip symptoms over a great many years.

The origin of pain in D.H.D. is not exactly known but some theories are; 1) arterial hyperemia with venous congestion leading to increased intraosseous pressure of the femoral head, 2) muscle spasm and contractures of muscles around the diseased joint, 3) joint capsule particularly sensitive to tension and tractions, 4) synovium sensitizing adjacent tissues and 5) periosteal irritation[9].

The patient plagued with hip pain will attempt to alleviate the pressure load on the affected hip joint. Figure 1 demonstrates the effect of the abductor force (M) and body weight (K) on compressive hip joint forces. If one tilts the trunk towards the painful hip, the center of gravity shifts laterally toward the femoral head, thus shortening the lever arm (b_2).- Therefore, the body weight (K) force decreased thereby decreasing the force (M) required by the abductor muscles. Hence, the total compressive force (R) on the hip is reduced.

A patient with a painful hip will sway or lurch during gait toward the affected side to decrease the pressure on the joint. This is called Trendelenberg gait. Over a period of time the abductors undergo disease atrophy. Therefore, if a patient with a chronic painful hip stands on the foot of the affected side their pelvis tilts downwards on the contralateral side. The downward pelvic tilt on the opposite side is called Trendelenburg sign. Other causes of Trendelenburg sign are: a) neurogenic disease such as poliomyelitis, b) neuromuscular disease such as myasthenia gravis and c) altered biomechanics eg. coxa valgus (causing the abdurator lever arm to shorten, b_1) in congenital hip disclocation.

A "axalgic" or painful hip gait is characterized by both Trendelenburg gait and "antalgic" gait. Antalgic gait is shortening of the stance phase during gait on the affected side. The secondary strain on the lumbar spine caused by this lateral lurch produces backache. Severe backache may also result from the compensatory lordosis which follows flexion contracture and functional shortening of the affected limb due to the adduction contractures.

Radiography

The usual order of joint changes seen radiographically with D.H.D. are:

(1) subchondral sclerosis
(2) narrowing of joint space
(3) osteophyte formation
(4) subchondral pseudocysts
(5) migration or displacement of the femoral head in relation to the acetabulum.

Symptoms do not necessarily parallel the degree of degenerative changes. Severe D.H.D. may exist with little or no pain and with good stability in spite of limited motion and deformity. Some cases of D.H.D. have been shown to spontaneously improve both symptomatically and radiographically in hips followed over fourteen years[10]. This improvement may be related to the formation of the fibrocartilagenous layer over the eburnated bone responsible for the reconstitution of joint space seen radiographically.[10]

Management

(A) NON-SURGICAL:

Non-surgical treatment is aimed at decreasing pressure loads on the joint and thereby allievates symptomatology. It includes:

- adequate daily periods of rest;
- avoidance of trauma or overuse;
- reduction of weight where indicated (3 lbs. reduction in static hip joint force occurs for each 1 lb. reduction in body weight),
- orthopaedic appliances: canes, crutches, walker; Figure 3 illustrates the effect of a cane on hip joint forces. Because of the long lever arm (b_3) at which the cane force acts a small force exerted through the cane will have significant reduction of hip joint forces: thus a cane used in the hand of the unaffected side provides 8-10 times decrease in compressive forces. Ten to twenty percent of body weight is usually placed on the cane which is equivalent to reducing hip load forces during gait to two times body weight[3].
- physical therapy: heat and massage to overcome muscle spasm, traction in abduction to stretch capsule and adductors, range of motion exercises;
- analgesics;
- intra-articular injection of steroids (1-2 injections at maximum and only in elderly patients).

(B) SURGICAL MANAGEMENT:

When pain and loss of function cannot be improved by non-surgical management, surgery is indicated. There are two broad categories of surgical therapy:

1) Joint Repair Without Using Implants

Therapy is directed at restoring the physiologic equilibrium by reducing the articular stresses. Theoretically two approaches are available but practically only one is useful.

a) Reduction of Load when Articular Surfaces are Congruent: Articular stress can be reduced by decreasing force R. This can be accomplished by reducing the magnitude of its component force M by a muscle release procedure. This technique known as the Voss hanging hip, was performed in the early years of D.H.D. surgery and was moderately successful at relieving pain, but does not give good functional results.

b) Reduction of Load when Joint is Incongruous: Physiologic equilibrium can be restored by increasing articular surface contact area with either a valgus or varus osteotomy. The rationale is that either method acts by reducing the compressive stress in the joint and redistributes the forces to a larger area or to a healthier area.

b-i) Femoral Varus Osteotomy

This is performed when varus displacement of the femoral head will increase joint surface contact (Figure 4a). A varus wedge ranging from 15 to 40 degrees is removed from the intertrochanteric region. The femoral head is displaced into

varus until joint congruity is restored. The adductors are released by tenotomy; the abductors and iliopsoas relax by proximal displacement of the greater and lesser trochanter. The distal femoral fragment is displaced medially to maintain the normal hip-knee axis[13] (figure 5).

In the early stages of D.H.D., the results can be seen in several months. The sclerotic subchondral bone in the supero - lateral acetabular roof disappears. It is replaced by subchondral bone of uniform density indicating uniform distribution or forces within the joint. The joint space widens, there is regeneration of fibrocartilage, pain is reduced and there is good range of motion and function.

Even in cases with severely destroyed joints, a varus osteotomy can be helpful. Radiologic changes are slower, yet the joint space widens, osteophytes disappear, and subchondral cysts heal[13]. Pain is reduced and the original range of motion is regained or slightly improved.[12]

b-ii) Femoral Valgus Osteotomy

This osteotomy is indicated when valgus displacement of the femoral head is required to produce a congruous joint and increase weight bearing surface area. For example, a varus deformity of the hip and a neutral or valgus deformity if there is a large medial-inferior osteophyte on the head in which it could support weight bearing forces. A lateral wedge is removed from the femoral shaft and the femoral head is rotated outward. The adductor and ilipsoas are tenotomized, and the abductors are relaxed by proximal displacement of the greater trochanter. The distal femoral component is displaced lateral to maintain the physiologic hip-knee axis (figure 5).

Bombelli advocates valgus osteotomy as the treatment of choice in most cases of D.H.D. suitable for osteotomy. He claims superior clinical results of longer duration, than with a varus osteotomy. This is based on the outcome of 1450 cases treated over 13 years. However a review of 2251 cases by the Swiss Orthopaedic Society found better results were obtained with a varus osteotomy. Pauwels claims that both procedures give equal therapeutic results provided joint surface area is increased, congruity is restored and operative indications are adhered to[13].

b-iii) Pelvic Osteotomy

Alterations in hip joint forces can also be achieved through pelvic osteotomies. The inominate osteotomy of Salter is used especially for congential hip dislocation.

Biomechanics of osteotomy:

1) Joint surface area is increased which reduces the compressive stress per unit area on the articular surfaces.

2) Muscle groups acting on the hip are relaxed. This has two effects: i) pain is reduced so that CG is shifted medially; ii) force M is reduced.

3) Femoral head is centered in the acetabulum.

4) Inclination of force R in the frontal plane is restored to 16 degree, Force R, and its components P and Q are restored to their normal orientations.

5) Magnitudes and directions

6) Muscle lever arm (b_1) is increased, and body weight lever arm (b_2) is decreased. Now force M can be decreased thereby reducing the magnitude of force R.

7) By dividing the femur, some authors believe the increased vascularity of the femoral head is reduced, accounting for the symptomatic improvement.

Indications for Osteotomy:[13] [12]:

1) Early D.H.D. with joint space narrowing, severe pain and functional impairment;

2) Young patient who will outlive total hip replacement;

3 Global-shaped femoral head;

4) Ability to radiologically demonstrate a congruous joint and increase surface contact; ie. improvement in adduction indicates a varus osteotomy;

5) Hip flexion greater than 70 degree preoperatively.

Varus vs Valgus Osteotomy:

Pauwels relies on pre-operative radiological assessment, to determine which type of osteotomy is indicated. Four views are required:

1) regular AP of pelvis showing both hips
2) neutral position
3) maximum abduction
4) maximum adduction

Taken with the hip internal rotated.

If joint congruity is restored by abduction, then a varus osteotomy is required, and if congruity is restored by adduction then a valgus osteotomy is indicated. When joint congruity cannot be achieved radiologically, both procedures are contraindicated, and other operations should be considered.

Advantages of Osteotomy:

1) Can be performed on younger patient with severe pain and disability whereas total hip replacement must be reserved for older patients.

2) Normal hip anatomy is maintained

3) Treatment is aimed at reversing the pathogenetic factors of D.H.D. and restoring the normal hip biomechanics.

4) Option for surface arthroplasty or total hip replacement (T.H.R.) remains.

5) The dreaded complication of the T.H.R. - infection is avoided.

Disadvantages of Osteotomy:

1) Surgical expertise in the procedure is mandatory. Most failures can be attributed to poor pre-operative assessment and operative technique.

2) Non-union of the two fragments is relatively common (10-15%) and is serious complication.

b-iv) Hip Arthrodesis

Fusion is rarely performed today because there are better surgical alternatives for treating D.H.D. and because most patients will not accept a stiff hip. In certain well defined cases, arthrodesis is valuable because a pain-free, stable joint results. It should be

considered in the young patient with severe D.H.D., who is active and places large demands on the joint. Certain criteria must be strictly adhered to:[18]

1) Age - should only be performed in young or middle aged person.

2) Condition of contralateral and adjacent joints - D.H.D. must be confined to one hip. The adjacent knee joints and spinal joints must be in good condition.

3) Status of Joint - joint should be severely destroyed, with very little range of motion, causing severe pain, and cannot be restored by osteotomy.

b-v) Girdlestone

This is an excisional arthroplasty in which an intertro-chanteric osteotomy is performed, and the femoral neck and head are removed. Soft tissue is interposed between the acetabulum and the femur. Its main indication today is for a failed T.H.R. in an elderly person, especially if failure is due to infection and reconstructive procedures are not feasible.[11] This procedure will reduce pain, restore motion, and correct contraction deformities. Its major disadvantage is that joint stability is compromised and the patient must use external walking aids and possibly a long leg brace to ambulate.

2) Joint Repair with Implants

a) Surface Arthroplasty

Surface arthroplasty resurfaces the acetabulum with a plastic cup and resurfaces the femoral head by placing a metal cup over top of it. Some modification of the acetabulum and femoral head is usually required to achieve a good fit with its resurfacing components. The long term results are still not known because it is a relatively new procedure.[1]

The two major indications are:

i) D.H.D. is beyond the stage where joint conserving osteotomies are feasible

ii) Younger patients (less than 60 years) who will out-live a total hip replacement

The advantages of Surface Arthroplasty over Total Hip Replacements:

i) If it should fail there is greater ease of conversion to alternative procedures due to the conservation of the femoral head and neck.

ii) There is no violation of the intramedullary cavity thus decreasing the risk of infection.

The major disadvantages of cup arthroplasty over total hip replacement are:

i) greater tendency to loosen

ii) less reliable in relieving pain

iii) longer rehabilitation program.

b) Total Hip Replacement

Total hip replacement (T.H.R.) replaces both sides of the joint with prosthetic components. Unlike, surface arthroplasty, the femoral head and neck are replaced and a metal medullary stem is inserted into the upper femur. Charnley who did much of the development of T.H.R. succeeded in creating stable artificial weight-bearing surfaces with low friction between components. The development of a low friction arthroplasty and the introduction of self curing cement represent the most significant achievements of orthopaedic surgery of this century.

b-i) Biomechanics

The estimated load on the femoral head in the stance phase of gait is equal to the sum of the forces created by the abductor and body weight and is at least three times the body weight. Therefore, total hip components must withstand chronic cyclic loading of normal activity and at times more strenous activity. These daily forces are in the range of three to ten times the body weight[7].

In coxalgic gait the CG is moved laterally toward the affected hip causing the length of the lever arm (b_2) of the body weight to decrease. The ratio of the lever arm lengths, b_1 -b_2, may now approach 1:1. Theoretically, this will decrease the total hip load by 30%[7]. Surgically, this ratio of 1:1 may be achieved by displacing the center of the hip medially by thinning the base of the acetabulum so that the acetabular component can be moved medially. Concomittantly, the greater trochanter is displaced laterally so as to increase the lever advantage of the abductors. Also, the abductor lever arm may be lengthened by increasing the length of the prosthetic femoral neck.

b-ii) Biomaterials and Fixation

There are two types of component combinations:

1) metal on metal (no longer popular)

2) metal on plastic (high density polyethylene) - acetabulum cup is plastic.

The three most commonly used metals:

1) stainless steel

2) chromium alloys

3) titanium

The charnley prosthesis, one of the most commonly used prosthesis is a stainless steel femoral head in contact with a high density polyethylene acetabular component. Fixation is achieved by self curing acrylic cement (polymethyl methacrylate). Acrylic cement should not be considered an adhesive glue because it possesses no adhesive properties. It acts as a filler which interdigitates with bone thereby eliminating the need for an accurate fit between the prosthesis and bone. This allows transmission of the load evenly over a fairly large area between the prosthesis and the inner surface of the bone.

b-iii) Biological Tolerance

There has been no evidence of carcinogenic or major toxic reactions to acrylic cement. At the bone cement interface there is a fibrocartilagenous layer of tissue which is an excellent medium for the transmission of forces. This provides a gradual transition in tissue elasticity which lengthens the life of the T.H.R.

Charnley's low friction arthroplasty has three main characteristics; 1) small femoral head, [2]2) lateral displacement of the abductors, and 3) medial

displacement of the center of the hip joint. The aim of low friction arthroplasty is to reduce the force load on the hip joint and prosthetic components so as to decrease the risk of prosthetic loosening.

The frictional force between the metal and plastic surfaces is constant for a given load. Since, frictional torque force is a product of frictional force times the length of the lever arm, it can only be reduced by decreasing the diameter of the prosthetic head. (Fig. 6) Frictional torque force is produced when the loaded hip moves through an arc of motion. During the same arc of motion, the distance travelled by a point on the surface for the small head is less than the large head.

Result:

Charnley has reported follow-up results in 106 T.H.R. over a period of nine to ten years[4]. Ninety-two percent showed complete success with no evidence to suggest that deterioration would be likely in future years.

The four major causes of T.H.R. failure are; 1) infection, 2) dislocation, 3) loosening of components and, 4) fracture of the prosthetic femoral stem. Deep infection after T.H.R. is often catastrophic because it is painful, disabling and costly, and usually requires removal of both components and cement. Therefore, a pre-existing infection of the hip, especially with gram-negative micro-organisms is one of the few contra-indications of T.H.R.

Summary

The importance of understanding hip biomechanics in relation to the pathogenesis, physical findings and management of D.H.D. has been stressed. The advancement of total hip replacement has produced significant pain relief in this common crippling disease. However, restricted activity and the use of the cane should still be encouraged in the management of D.H.D. □

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Review of Epithelial Wound Repair

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Literature dealing with wound repair and related topics appears periodically in various scientific and medical journals. Recently, many developments in this field have been presented, but with the understanding that previous articles have been read. This paper highlights the more interesting new findings in wound repair and attempts to provide such background information as will induce an appreciation for these recent advances.

Any wound represents a traumatization of tissue, with or without an interruption in continuity. This is remedied, to a certain extent, by all human tissue. However, the ability to regenerate is best demonstrated by the heading of an epithelial wound. This is of concern in all surgical specialties, where an inscision through the skin is common in most operations.

It was Gillman[6] in 1955 who initially compared the old ideas with respect to the sequence of healing, with the ones now accepted as correct. Gillman sought to prove that:

a) A blood clot is not essential in the règgenerating sequence since it does not act as a biological scaffold for the epithelial cells,

b) The dermis does not regenerate and form granulation tissue prior to epithelialization,

c) Fibroblasts do not originate from blood borne precursors.

Recently, much research in the repair of wounds has revealed unique cell migration and transformation; both of which are necessary for total tissue regeneration and repair.

An examination of the initial response, epithelialization, dermal healing (including fibroplasia), contraction, infiltration by myofibroblasts and intercellular material, as well as some nutritional aspects will provide a basic understanding of the process of wound healing and an appreciation of recent advances made in this field.

Initial Response

When experimental and accidentally formed wounds were studied it was observed that an inflammatory reaction occured first. Initially, the dermal edges of the wound increase in cellularity, with a high influx of polymorphs, eosinophils and monocytes. The tissue with the highest concentration of leukocytes demarks the necrotic area, which extends to include all the structures 100-200 mm from the edge of the wound. These early migratory cells are replaced within 48 hours by macrophages, lymphocytes, and fibroblasts. Fibroblasts must be present and activated before dermal collagen and elastin regeneration can take place.

An activator substance, produced by the macrophages, is believed to initiate the regeneration process[5]. Production of this stimulatory factor is inhibited by the anti-inflammatory action of cortisone and related anti-inflammatory drugs. Therefore, the primary response of the body is defense against possible infection.

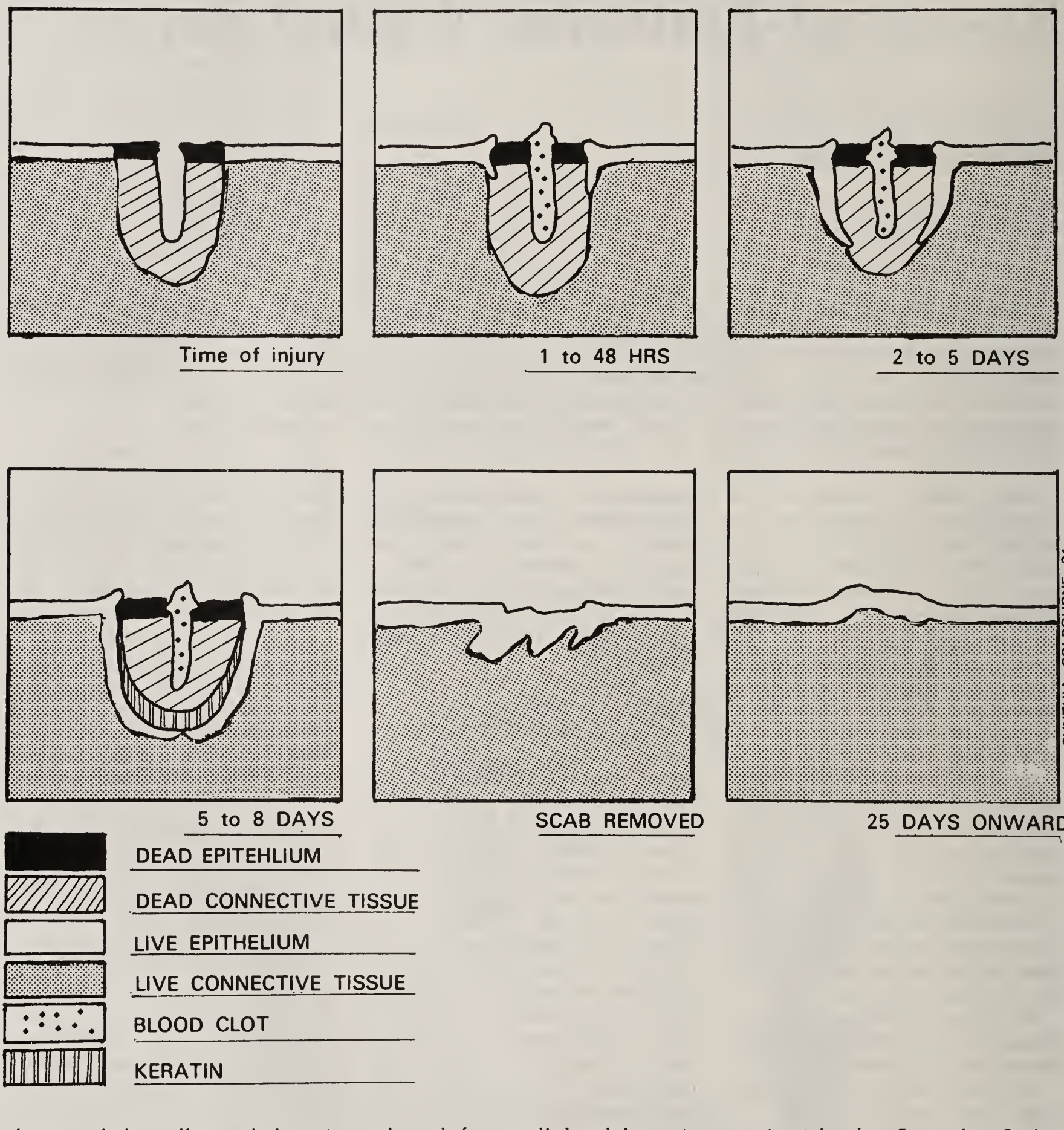
Epithelialization

Once the initial response has established a suitable environment, epithelialization may commence. Within 24 hours post trauma, epithelialization begins at the level of the stratum basale and stratum spinosum. The mitotic activity of the basal layer is stimulated by lack of contact inhibition at the wound edge. A 'heaping up' of epithelium occurs at the outer edge of the necrotic area initiating migration of epithelium in the form of two spurs. In excised wounds, however, epithelial migration from the edges is accompanied by additional migration from sweat glands and hair follicles. Cells of the stratum spinosum undercut the necrotic area and moving on a thin layer of fibrin, which guides the cells, circumscribe the entire blood clot.

How do cells of the stratum spinosum migrate if attached to surrounding cells by spot desmosomes? This problem is overcome by the dissolution of those desmosomes surrounding the wound. Croft[1] et al. observed that there is an enormous decrease in these cell to cell junctions at the start of migration. Nevertheless, the epithelium moves as a unit, thus contrasting significantly with the migration of carcinogenic epithelium, where the cells migrate individually.

As the leading spurs progress

Epithelial and Dermal Regeneration in Wound Repair



downward the cell morphology is altered dramatically. The cells develop contractile elements, become phagocytic and increase their lysosome content. The invasiveness of the epithelium is aided by degradative enzymes of disputed origin. Many researchers believe that the enzymes are released from cell breakdown in the area, while others argue that the enzymes come directly from epithelial cells.

Within 5 days epithelialization is complete. The leading migratory edges have contacted each other allowing for the re-establishment of contact inhibition. From approximately day 5 to day 8 the fibrin layer is removed and a new basement membrane is formed. This is a time of epithelial hyperplasia. Psuedopods are extended from the epithelium into the dermis and act as anchors for the newly regenerated tissue. Contact with the dermis is believed to slow

epithelial penetration and has been compared to the decidual reaction in maternal connective tissue during the implantation of the ovum. Hyperplasia does not occur equally within the excised wound; instead a blistering phenomena takes place between the fourth and sixth day. During this period, the epithelium is anchored only by its outer edges and one must exercise caution in changing dressings to avoid reinjuring the extremely delicate epithelium.

Since keratinization can now commence the blood clot can easily be excised. If left on the wound, the clot would slow dermal fibroplasia and due to its flexible nature increase the possibility of unsightly scarring. With the avoidance of epithelial reinjury and the excision of the blood clot, epithelialization is complete and the next stage of wound healing, fibroplasia, can begin.

Dermal Healing

Fibroplasia

Dermal healing involves fibroblast proliferation, collagen and elastin production, and reorganization. No collagen synthesis is evident before the third day post trauma. It is only after epithelialization has been completed that dermal regeneration takes place, (4-6 days). There is a general increase in fibroblast activity, and synthesis of all intercellular substances of the dermis, including mucopolysaccharides, collagen and elastin.

The origin of the wound fibroblast has recently been determined conclusively. Russell et al, employed parabiotic rats (rats sharing the same circulatory system). He radioactively labelled the leukocytes of one rat and experimentally wounded the other. No trace of radio activity was found in the fibroblasts of the wounded rat, thereby disqualifying the majority of research into the transformation of blood borne cells to fibroblasts. It is now believed that the invasion of the dermis by fibroblasts originates from adjacent undamaged connective tissue. The fibroblasts are derived from primitive mesenchymal cells. Pericytes and cells from subcutaneous fat, if affected, and nearby fibrocytes are the believed precursors. Fibroblasts represent a major regenerative component of the granulation tissue in the production of intercellular substances.

New capillaries begin to bud off from the endothelial cells of preexisting capillaries. They vascularize the dermal area causing the wound to 'puff out' and turn pink. This stage continues until approximately the twentieth day post trauma. Granulation tissue, which consists of vascularized connective tissue, has now been formed and possesses many unusual properties. With the increase in collagen and other intercellular material, the cellularity of the region begins to decrease on day 10 and levels off by about the third week.

After this initial phase of fibroplasia, there is a rise in the tensile strength of the wound due to the reorganization of the regenerated tissue. Usually, by day 15, the collagen production has levelled off, but the amount produced never exceeds that of adjacent undamaged tissue. Subsequent to the reorientation of the epithelial cells there is a realignment of the spindle shaped fibroblasts into the plane of highest stress. Similarly, but in the late stages of healing, the collagen and elastin produced become oriented parallel to the skin surface. By day 84 the majority of fibroblasts have assumed the fibrocytic form and cicatrization is virtually complete.

This sequence of events pertains to any type of epithelial wound repair, and any deviation from it represents a possible anomaly in the healing process. With this knowledge of the progressive stages of wound repair, many clinical applications have been developed. A few of the more interesting ones are mentioned by Gillman[6] et al, and Lindsay[7] et al. They consider methods of bandaging incised and excised wounds as well as the healing of areas from which split skin grafts are harvested.

Lindsay[7] et al describe a simple suturing technique for incisional wounds that combines careful opposition of the wound edges and the minimization of clotting to insure quick and complete healing.

Contraction

The phenomenon of contraction, or the self initiated closing of a wound, has been considered by many researchers. They believed it to be a form of inherent defense on the partof this tissue. It is necessary to distinguish between contraction and contracture, the latter being the development of deforming and/or disabling scar tissue. Although wound contraction is universal it may lead to a certain amount of contracture with an accompanying loss of normal function. Much research is now in progress to study the possibility of separating the beneficial properties of contraction from those which cause scarring.

Contraction may not be completely detrimental if the wound is distant from articulation. For instance, the body controls large abdominal wounds in this manner and precise studies have shown that after the incision, contraction begins in approximately 5 to 7 days. As a result the wound is reduced to as little as 30% of its original size. All full thickness wounds on a mobile epithelized limb and all internal organ wounds display this type of contraction, thereby becoming a problem for the surgeon.

Gabbianni[3] et al studied the characteristics of the granulation tissue produced in experimental wounds. At that time it was hypothesized that contraction was due to the realignment of connective tissue fibres by some unexplained force. It was Gabbianni who observed the unusual morphology of some of the fibroblasts and noted their striking similarity to smooth muscle cells. In addition he found that these cells had many features similar to mesenchymal

foetal cell morphology. Immunologically, the tests showed the presence of a functional amount of actin in the form of fibrillar intracellular bundles which allowed an interesting hypothesis. Gabbianni[3] et al proposed the existence of myofibroblasts, which combine the features of both cell types and are contained within the granulation tissue. From this work and hypothesis came many studies dealing with the control of contraction by pharmacological manipulation of the newly recognized myofibroblast.

The Myofibroblast

Myofibroblastic cells are observed after collagen regeneration has been established and vascularization is almost complete. Their contractile role becomes apparent approximately 7 days post wounding. At the same time fibroplasia of the dermis is at its peak. The myofibroblast has numerous cell to cell desmosome type junctions which are believed to be specialized for the redistribution of contractile forces from one cell to another, thus forming a mechanical syncitium. The nucleus shows many indentations and contains an extensive cytoplasmic fibrillar system. Pharmacological testing reveals many smooth muscle cell characteristics.

Madden[9] et al employed a smooth muscle antagonist, trocinate, to study the pharmacological characteristics of the myofibroblast and found equal results on both smooth muscle and granulation tissue. However, when the antagonist was removed, active contraction resumed. There was no accumulation of collagen or elastin, suggesting that the actual forces of contraction are contained in the myofibroblasts themselves and are not inherent in the connective tissue fibres. Similar research by Gabbianni[7] found that granulation tissue contraction could be induced by the same drugs that caused contraction in smooth muscle (i.e. 5-HT, angiotension etc.). Antihistamines also decrease the rate of contraction by occupying histamine receptors on the myofibroblasts. As yet, few clinical applications have been derived from these findings. Research continues to thrive in the field of reducing contraction in pathological conditions by drug therapy instead of the commonly employed surgical approach.

Many unusual and deforming diseases are caused by the presense of active myofibroblastic nodules. Two of these diseases are *Dupytren's contracture*, where the palmar aponeurosis is affected, and *idiopathic carpal tunnel syndrome*. In both cases the only available remedy is surgical removal of the affected tissue, but this does not resolve the problem of recurrence, and furthermore, inhibits the patient's easy return to normal function due to a extended post-operative physiotherapy. Myofibroblasts are not only common in wounds and other pathologies but also are normally found in duodenal villi, testicular capsule, lung tissue, and periodontal ligaments where their function is not fully understood. Certainly, a cell combining the features of two cells poses many interesting research questions that can only be answered with more study.

Collagen and Elastin

Studies by several researchers have recently revealed that elastin, previously considered unimportant in the repair of a wound, actually plays an integral part. It is essential in the development of contractile forces and the transferral of mechanical stress applied in the wound area, thereby maintaining the integrity of the newly formed dermis.

The study of collagen synthesis during wound healing led researchers to the discovery of sequential protein construction. Collagen is synthesized as a protein, hydroxylated, and then deposited externally as tropocollagen where it is transferred to its final periodic collagen form. It was also found that not all collagen synthesized during wound healing is composed of the same precursor proteins. Normally in adult skin Type I collagen is produced. The granulation tissue produces a Type III collagen similar to that found in embryonic skin and the aorta. This allows for a greater elasticity than that provided by Type I collagen. The regeneration of connective tissue is also important in scar remodelling. Here, newly formed collagen is reabsorbed by the fibroblasts and is deposited with altered orientation. The amount of collagen available never exceeds that already present in undamaged adjacent tissue, thereby producing scars of comparable strength.

Elastin has been a source of controversy for many years. It was thought to have little consequence in wound healing and to be absent until almost two months post wounding. Work by Shwartz[14] et al and Telfer[17] et al proved conclusively that elastin is produced by fibroblasts at about day 8 in concentrations greater than the surrounding non damaged tissue and lasts up to 150 days post wounding. It was also noted that myofibroblasts surround themselves with elastin and employ it as a biological tether.

Immunological research displayed elastin to be most abundant near the edges and in the centre of the wound. Also ascertained was the fact that in the wounding of tissue that normally contains no elastin (eg. the liver), it was still produced by the healing process. The analogy was drawn that the myofibroblasts acted as the muscle, and the elastin as the tendons to produce a contraction mechanism. This is the current concept of the functional relationship between myofibroblasts and elastin.

Nutritional Effects

Good nutrition is essential for the effective repair of wounds. Since all wound repair requires a certain amount of regeneration, the raw biological materials must be present in the correct place and amounts to allow proper tissue synthesis.

Lack of protein, (Kwashiorkor

syndrome) causes a marked decrease in the tensile strength of the wound. This is due to a shortage of both fibroblasts and collagen. In wound repair, methionine is a primary amino acid required in collagen synthesis. Although it represents only 1 per cent of all the amino acids in collagen it cannot be synthesized by humans. Methionine is the most important source of cysteine. This amino acid is not directly involved in collagen synthesis but the sulphated amino acids are thought to have a supporting role in various other cell mechanisms. Ascorbic acid is necessary not only for collagen synthesis but also for the maintenance of previously healed wounds. In the area of the wound the ascorbic acid concentration increases in proportion to the rate of collagen synthesis. With a lack of ascorbic acid the fibroblasts revert to inactivated fibrocytes. There is a build up of free ribosomes, a decrease in E.R., and an increase in stored fat. This leads to an overall decrease in protein synthesis and a halting in the repair of the wound. A deficiency of ascorbic acid (scurvy) is a source of major detriment for the synthesis and maintanence of connective tissue.

Summary

Recently many advances regarding the sequence of wound repair have been made. In order to fully appreciate these developments, a rudimentary understanding of wound repair is essential. The initial response to trauma, epithelialization, fibroplasia, contraction, myofibroblast proliferation, the production of intercellular substances, and an adequate state of nutrition are all factors which are necessary for wound repair. Developments made in the understanding of the importance of these factors have led to improved wound repair under various conditions. □

Acknowledgements

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Conclusions and Recommended Approach

When a patient arrives with an ankle injury, certain steps should be taken. The ankle should be examined and if a sprain is suspected, the relevant history and physical findings should be looked for and quantified. If the injury is then considered to be significant, a plain radiograph may be obtained to rule out an avulsion fracture and then a varus stress view can be taken of both ankles.

If there is a detectable degree (greater than three degrees) of difference in tilt between the two then the injured ankle should be casted, otherwise strapping is sufficient.

Finally, if after a period of time the function of the ankle is still not satisfactory, then surgery should be considered. □

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available on upon request

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The Antisocial Personality Disorder

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The antisocial or psychopathic personality disorder harbours different connotations and implications for various subgroups of our society. For the medical profession, it has presented a diagnostic dilemma ever since the characteristic behaviour pattern was described by Pinel in 1806. Pritchard (1831) applied the term "manie sans delire" or "moral insanity" to describe the typical psychopathic picture. In 1974 Sir Aubrey Lewis summarized the prevailing diagnostic confusion when he stated, "for 150 years the diagnostic concept called 'moral insanity' has been troubling psychiatric nosologists." To this day, psychopathy remains an imprecise descriptive label, largely dependent for its definition upon the standards of the clinician making the assessment.[1] Even if a set of well defined diagnostic criteria could be agreed upon, any deviation from the average is strictly a matter of degree, and many of these deviant personalities suffer no disability, presenting no problems for society. For the public, the psychopath represents an individual involved in the most notorious crimes of sex and violence. For the legal profession, psychopathy presents the question of hospitalization vs. incarceration, with the underlying responsibility to protect society from a potentially dangerous offender. Although over the years our diagnostic criteria have become more refined and our insight into the developmental factors has improved, very little progress has been made in the treatment of this disorder.

Clinical Features

The antisocial personality disorder characteristically onsets prior to age 15 and persists into adulthood. Prominent childhood symptoms include: lying, fighting, stealing, truancy from school and resisting authority.[2] Behaviour is unsocialized and very hedonistic. Enuresis is occasionally seen. In adolescence, early or aggressive sexual behaviour, alcohol and illicit drug abuse, poor academic achievement and resistance of the familial value system are commonly seen[2]. Males typically manifest the behaviour patterns at an earlier age, as well as more frequently than females, Male: female sex ratios as high as 8:1 have been reported.[2,3] Evidence for racial differences in incidence has not been forthcoming. Although the psychopath demonstrates markedly deviant behaviour patterns, defects in intellect and reasoning are rarely observed. As adults, sexual deviations are common and there is an increased tendency towards alcoholism and illicit drug abuse. The clinical profile of antisocial behaviour is outlined in Table 1.

Psychopathy is primarily a defect of the emotional life, typified by the inability to experience normal human emotions. Superficially, these individuals often appear likeable and charming, while underneath they are cold and callous. They are extremely manipulative, and their actions are unpredictable and impulsive, being driven by primitive drives and hedonistic urges. Emotional immaturity is frequently evident. The capacity to learn is impaired, and consequently, the psychopath benefits little from adverse experiences or punishment. Antisocial personalities have an impaired capacity to maintain lasting responsible relationships with family, friends or marital partners, and fail to sustain satisfactory job performance over substantial periods of time. The psychopath is not loyal to any group or individual - he has a completely hedonistic and egocentric approach to life.

Morals and ethics mean little to these individuals and they are unable to conform to societal demands or the prevailing cultural milieu. They show a lack of regard for the consequences of their actions and are consequently often chronically in conflict with the legal authorities. The psychopath has little if any future orientation to life[4], and therefore his actions are guided by immediate gratification. He generally has little if any insight into his condition. When challenged with the consequences of his actions, he has little inclination to do anything other than rationalize them in an effort to avoid all personal responsibility. This rationalization frequently contains fabrications and distortions of reality, and is rarely a factual account.

Neuroses are frequently seen in antisocial personalities.[2 3 5] These individuals are predisposed to anxiety reactions because their actions lead them into conflict where they themselves must suffer the consequences. Surgery is oc-

Table 1*

Clinical Profile of Antisocial Behaviour

Superficial charm and good intelligence
Poor judgement and failure to learn by experience
General poverty in major affective reactions
Unresponsiveness in general interpersonal relations
Pathological egocentricity and incapacity for love
Absence of delusions and other signs of irrational thinking
Inadequately motivated antisocial behaviour
Lack of remorse or shame
Untruthfulness and insincerity
Specific loss of insight
Unreliability
Failure to follow any life plan
Suicide rarely carried out
Sex life impersonal, trivial, and poorly integrated
Fantastic and uninviting behaviour with drink and sometimes without

* Modified from Winokur, G. and Crowe, R.R. "Personality Disorders." in Comprehensive Textbook of Psychiatry. Ed. 2. A.M. Freedman, H.I. Kaplan and B.J. Sadock (Eds.) Williams & Wilkins, Baltimore, 1975. p. 1291

casionally used as a means of releasing anxiety and some psychopaths crave and have experienced multiple operations. These individuals conform to the Münchausen Syndrome[6]. Psychopaths frequently make suicidal threats but only rarely commit the act, using these threats as a means of environmental manipulation. A small minority of antisocial personalities are seen in medical clinics, with a far greater proportion being encountered in penal institutions. Recent studies estimate that between 20-78% of male felons and 65% of female felons could potentially be classified as psychopathic.[2]

Many antisocial personalities lose their psychopathic nature at advanced age. Several investigators have noted that the natural history of violence, criminal and antisocial behaviour, decreases after age 40.[2 3 7] Reid[3] found that 35-40% of psychopaths "burn out" at a mean age of 35. Tucker and Pincus[2] have proposed that as a rule of thumb, "after 21 years of age, 2% of all antisocial personalities remit (grow up) each year."

Diagnosis

Unlike the majority of psychiatric conditions, mental status examination is of greater value in excluding the diagnosis of antisocial personality than in establishing it. Consequently, the mainstay of diagnosis is the history as elicited from the patient and immediate family members. In assessing the antisocial personality, it must be noted that deviant behaviour is an inadequate diagnostic standard. Although impulsivity, aggressiveness, and egocentricity are symptomatic of psychopathy, they do not necessarily distinguish it from other deviant behaviour patterns. All criminals are not necessarily psychopaths and all psychopaths are not necessarily criminals.

The initial step in the management of psychopaths, as in all other medical conditions, is to investigate and subsequently negate all treatable causes potentially responsible for the observed behaviour. In this regard, conditions such as alcoholism, drug abuse, manic phase of cyclothymic personality disorders, schizophrenia (especially in childhood), temporal lobe epilepsy, cerebral trauma, mental retardation, and encephalitis, must be considered.[2] Once these have been disregarded, one must exercise caution differentiating between primary conduct disorders, aggressive or criminal behaviour, dyssocial behaviour and the antisocial personality. Finalizing the diagnosis carries with it a grave prognostic outlook and offers little in the way of beneficial treatment possibilities.

Etiology

Psychologic theories of etiology have concentrated on two major possibilities - the development of conscience and the quality of the parental experience, especially the mothering.[2] Bowlby[11] felt that psychopathy was related to maternal deprivation in the first five years of life, and reported an increase in disrupted affective bonds during the childhood of individuals who were subsequently diagnosed as psychopathic. Cameron[12] reported that "if the significant parents are capricious, inconsistent or emotionally neglectful, the child may grow into adulthood with irresponsible or emotionally shallow and meaningless attitudes toward others." A number of other investigators have identified additional factors which include: childhood rejection[13], early loss of a parent or surrogate of the same sex[4,12,13], head trauma[2,7,13], low socio-economic status[2], poor educational level[15], coming from a broken home[2], and city *vs* country dwelling[2]. These experiences are often present in the history of a

psychopath, and possibly are related to etiology though none have been identified as causal.

The psychoanalytic postulates of etiology are expressed in the writings of Sigmund Freud. Freud insisted that the psychopath was the product of a traumatic disturbance during the oral stage of psychosexual development. As a consequence of infantile sexual trauma, a resultant inadequate development of the ego and superego ensued. Hence, the psychopath is an individual whose behaviour is largely determined by id impulses.

Twin studies conducted by Mednick[8] found a 60% concordance in monozygotic twins and 30% concordance in dizygotic twins bearing a diagnosis of antisocial personality. Studies on 47 XYY and 47 XXY individuals[2, 9] have produced conflicting results, and hence fail to conclusively associate behavioural disorders with these genotypic configurations. However, on the basis of twin studies, and the fact that there is a prominent genetic component to personality, it appears that a tendency to antisocial behaviour has a genetic basis, although additional (environmental) factors are also operant in the genesis of this condition.

In searching for an organic basis for this disorder, various researchers have employed the electroencephalogram (EEG). Hill[10] described electroencephalographic changes, indicative of defective cerebral maturation, in greater than 50% of individuals diagnosed as psychopathic. The most common abnormality was excessive bilateral rhythmic slowing, showing theta waves mixed with dominant alpha activity, associated with prolonged slowing after hyperventilation. Such abnormalities were detected in the temporal lobes of over 80% of these individuals.

The above points raise a number of interesting possibilities in the etiology of psycopathy, although to this date, no single factor nor group of factors has specifically been identified as causal in the genesis of this disorder.

Treatment

Therapeutic pessimism surrounds the antisocial personality, as it presents one of the most difficult treatment problems in psychiatry. In fact, a number of authorities believe that it is hopeless to attempt to treat these individuals.[1, 2, 3, 12, 14, 15] Cameron[12] found that the major obstacle to change was the patient's own attitude, especially when he insisted that there was nothing wrong with him, he didn't need help and refused to accept it. Institutionalization has generally failed to provide a cure and punishment is impractical due to the psychopath's inability to learn from experience. In practice, dangerousness often becomes the key consideration and is consequently the basis on which many therapeutic decisions are made. In dealing with these individuals, a very important decision is establishment of the point at which further medical intervention would fail to be efficacious. At this stage it is in society's best interests to have the deviant behaviour dealt with by the due process of law under the prevailing legal system. Ethical difficulties are inherent in dealing with these individuals. The Baroness Wootton of Abinger expressed this in the 53rd Maudsley lecture (November, 1979) when she said, "Psychiatrists can often alleviate the miseries of the mentally ill, and assist the 'maladjusted' to attain their own goals or to conform the society's demands, but in so doing they may have to face ethical problems affecting both their objectives and their methods of treatment particularly where the convicted offenders are concerned."

Psychotherapy has been very disappointing as a treatment regimen. Psychopaths lack the desire for change and anxiety over their condition, which most therapists regard as prerequisites for treatment. Additionally, the efficacy of psychotherapy is dependent upon the establishment of a therapeutic relationship, and the psychopath is generally incapable of establishing meaningful therapeutic rapport. The therapist must constantly be aware of the manipulative attempts of his patient, must stick to his judgements, and resist attempts by the psychopath to alter his advice. When dealing with psychopathic females, their feelings of love, including sexual, are less controlled by reason and consequently the therapist must be wary of attempts to foster sexual improprieties. If a therapeutic relationship is established, rational guidance and affection, with firm but not aggressive rules, should be employed to direct the client. Caution must be exercised when confronting these individuals, as their reduction in foresight and impulsivity predisposes to violent outbursts (sometimes physical) which may be directed towards the therapist.

A question which frequently arises during the treatment of these individuals is - Is the psychopath legally responsible when he commits an offense? This issue is of grave importance to both the medical and legal professions. Under several judicial systems (including the Canadian), the policy of relieving a mentally impaired individual of responsibility has been outlined in the McNaughten Rules. Daniel McNaughten was a paranoid schizophrenic who killed William Drummond, the Parliamentary Private Secretary of British Prime Minister Sir Robert Peel in 1843. McNaughten mistook Drummond to be the prime minister and shot him while under the delusional belief that only by shooting the prime minister could he be relieved of his persecution. This resulted in a set of rules being established by the judges in the House of Lords that have served as an index of responsibility ever since. The two rules for determining the responsibility of an individual who pleads insanity in defence of a criminal offense are as follows:

1. To establish a defence on

the ground of insanity it must be clearly proved that, at the time of committing the act, the party accused was laboring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing; or if he did know it, that he did not know he was doing wrong.[16]

2. Where a person labors under partial delusions only and is not in other respects insane (and commits an offense in consequence thereof) he must be considered in the same situation as to responsibility as if the facts with respect to which the delusion exists were real[16].

Ever since their initial formulation, these rules have been repeatedly criticized and attempts made to change them. It has long been recognized that responsibility is not an all-or-none phenomenon, but is present in varying degrees. In addition, the McNaughten rules emphasize the rational and cognitive elements in determining behaviour, without consideration of the emotional forces involved. They also require the psychiatrist to give opinion on the question of responsibility, something which is best left for the judge and jury to decide. Gunn (1977) summarized the current situation when he said, "In British courts in these days questions of responsibility have largely given way to questions of the type and degree of any mental abnormality present, its treatability, its possible mitigating effects on the sentence, and the management problem presented by the offender."

Several investigators have concluded that very few possibilities exist for the pharmacological treatment of psychopaths. [1, 2, 3, 14, 15, 17] Antisocial personalities are refractory to psychotropic agents. [14, 15, 17] Previous claims that pericyazine or propericiazine (both phenothiazine derivateves) reduced antisocial behaviour have been unsubstantiated by controlled trials. The individuals who benefit from pharmacological treatment are those whose predominant symptoms are uncontrolled aggression, impulsiveness and lability of mood. Lithium is promissing in this regard, as it has been shown to reduce the tendency to aggression and violence in violent offenders.[3] Since some of the behavioural reactions are attempts to prevent the development of manifest anxiety, recent studies suggest that minor tranquilizers may prove beneficial in this respect.[14] However, the danger inherent in this approach is that consequent to the constitution of the psychopath, serious drug dependencies may result.

Psychodrama has proven beneficial, in that role playing helps show the psychopath the feelings of others and thus makes him better able to maintain affectional relationships.[15] Electroconvulsive therapy (ECT) fails to alter the personality structure and hence has no effect in the majority of cases.[15] A small proportion of individuals respond favourably to hypnoanalysis[15]. Group therapy appears promising, because it provides the opportunity for self-confrontation. In groups, psychopaths are more apt to be honest with one another, than with the therapist alone.[2, 3, 5] A number of authorities have noted that the natural history of antisocial behaviour seems to decrease after age 40.[2, 3, 7] Therefore, due to the poor results obtained with the aforementioned treatment regimens, one way of dealing with these patients would be to isolate them from society until after age 40 or until they have "burned out."□

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"Hello, I Must Be Going": a note of dying

Harley Hahn 8T4

"So he begins again, calmly explaining to the doctor that the world is sad only because we imagine it that way, and that the way to change the world is to change the way we imagine it. He says the Pesseemans are living in a novel in which death is the worst thing that can happen. But that in fact there is no more reason to think of death as bad than as good. The question is completely open. So it's best to see death, and life, in a way that makes you happy in the present. And this better novel is the planet Opteema. He claims it is this pessimistic view of death that leads to a futile grasping at life and possessions, and ultimately, to war."

"It's OK to live on Pesseema if that's what you want to do...But when you die, you gotta switch to Opteema...You gotta, Jerry...Death is too terrible on Pesseema...You need to believe in magic when you're gonna die..."

Carmen Miranda Memorial Flagpole
by Gerald Rosen

"There is meaning and order in all things--
stop and smell the roses."

Health is a Question of Balance
by Paul Brenner

You are scared of death — scared in a peculiar way. It has an awesome charm all it's own. There are sporadic moments that come to you when you glimpse the shadow of your death. A profound, even terrifying feeling sweeps your mind. Here is something that is programmed so deeply into you that its tendrils wrap around your very existence. Death is a part of you as surely as is life.

I had the pleasure recently of attending a meeting of health practitioners at the Mount Sinai Hospital in Toronto. These meetings, organized by Dr. David Shaul, have been taking place on a biweekly basis, off and on for five or six years. The players change but the theme is the same: to explore the needs and feelings of patients, and the doctors, nurses, and social workers who serve them. The topic this week was death and the atmosphere was warm and encouraging as they discussed the imminent death of a patient. They talked about death in a hospital: how do you tell someone that they are going to die? How do you coordinate the efforts of the doctors, nurses, social workers, and the family?

I listened as they talked and I noticed that they assiduously avoided two important questions. First, they all agreed that it is a difficult job to inform someone that they are going to die. But why? Why out of all the bad news that a doctor is forced to give is this his/her saddest job? Why does it affect us so strongly? And second, what about helping the terminally ill? What do these health practitioners want to accomplish? What are their goals?

Why did these concerned intelligent people unconsciously avoid articulating these questions? Could it be that when humans enter the realm of death they are faced with a force so terrifying, so powerful, that the mention of it out loud is cutturally forbidden?

Let us examine the transition which ends our lives and what it means. You will find, strangely enough, that as a human being you are caught in an enormous cosmic paradox. A paradox in which life is meaningful chiefly because of death. Even more peculiar, you owe your appreciation of life to the fact that your predecessors died and that you too, will one day cease to exist.

If there is one characteristic that describes terrestrial life it is the ability to adapt to change. In fact, we could use that as a definition of life itself: an entity that can adapt itself to changes in its environment is alive.* The ability to adapt is probably the most important characteristic an organism can have on our planet. Indeed, the better a species is at adjusting to change, the more successful it is; that is, the better its members can reproduce and survive.

The ability of a species to survive lies in its large number of members. Although each member can only respond successfully to a limited amount of change, the combined potential of all the members is great enough so that some of them will (hopefully) survive anything.

Viewed in this light, a species takes on a life of its own. That is, we can consider a species to be a large organism made up of individual members much as we consider an animal to be made of in-

*There are, of course, more robust definitions of life. For instance, some people feel that to qualify as being alive an entity must be able to reproduce itself (at least with the help of a partner). If you believe this to be so, you might ponder over hybrids - like mules - which cannot reproduce although they are certainly alive.

dividual cells. Although, each cell does its job reacting to its environment, seemingly oblivious to all the other cells, we — as outside observers — can recognize the ggeneral scheme of things — the whole organism.

As an exercise in thought, remove yourself from the Earth and look down on all the human beings at once. Now imagine the sum total of all the humans throughout the entire lifetime of the species (so far). Viewed in this way, we can see a remarkable organism. An organism of which we and all our ancestors form a part. A life-form like this is so important as to demand a name so let's call her* Terra, after her birthplace.

Terra has essential differences from the organisms we are used to. For one thing, her cells (humans) are spread out over space. However, just as our cells are bathed in interstitial fluid which provides nutrients and carries away wastes, Terra's cells are surrounded by a terrestial environment that does the same.

Terra is thought to have developed about two to four million years ago. In the last ten thousand of these years she has undergone a change in consciousness allowing her to exert a remarkable level of control over her surroundings. In recent times, Terra has sent several of her cells into space and to the moon, and it seems altogether likely that she will begin to explore away from her home planet. After all, the Sun has a limited (though long) lifespan; if Terra is to survive indenfinitely she must ultimately emmigrate.

It is important to understand something of Terra's nature. Although our personal relation to life and death seems paradoxical and without meaning, Terra's relationship to the life and death of her cells makes sense.

Consider Terra's cells, which I will call *Terracytes*. A terracyte has a limited lifespan — it is a rare one that lasts a hundred years. It is important for Terra's well-being for the terracytes to look out for their own self-interest. They must be programmed to want to live as long as they can.

This in itself is not unusual for a lifeform. All organisms and cells, with only the occasional exception, seem to try to live as long as possible. The unusual fixture of terracytes is that over the course of their evolution they have developed a sufficient degree of sentience and of self-awareness to be aware of their mortality.

The Eternal Human Conflict: your mind, your conciousness, wants to live forever; but your body is programmed to die. And the great cosmic joke of it all is that *you know it*. You have to appreciate black humour to laugh at this one, but it is funny — probably the funniest thing that has happened in the last hundred thousand years.

As a species, Terra is nothing if not successful. She is composed of terracytes that afford her enormous potential to adapt — producing offspring that are slightly different from themselves, and then dying. The important point is that the programmed death of all the terracytes is necessary. As far as Terra is concerned, immortal terracytes would soon overburden her and her home with a plethora of useless cells.

But where does that leave you? It is fine to consider how your birth, mating, and death are good for Terra; but you live inside *your* mind/body, not hers. Consider that although your individual cells seem to work together in your interest, this is an illusion. As far as one of your cells might be concerned — if it could think — it is looking out for its own self-interest. Fortunately for you, your cells have evolved so that under favorable conditions their collective self-interest is your self-interest.

In a similar way, individual terracytes may spend their lives looking out for themselves and their neighbours and still, in the long run, serve Terra's interest. Perhaps it is unimportant for terracytes to be aware of this, just as it is not necessary for your individual cells to be aware of their collective work. But your death is not a capricious cosmic whim it is both useful and necessary.

So why is it difficult for doctors to tell their patients that they are going to die? Think of it in terms of yourself. You perceive the time after your death as a vast unknown and you are afraid of that unknown. You have a mind that wants to live forever, living inside a body that will die. Confronting someone else's death reminds you of your own mortality and lifts the veil from that fear.

As is usual where the "disease" is due to ignorance, the relief comes from enlightenment. Instead of avoiding the thought of your death, seek it out and explore it. See yourself as more than just an aimless pinball being bumped hither and thither until the game is over. At the very least, you are part of a species-organism whose elegance is compelling.

And the second question: What can we hope to accomplish by helping those that are dying? You can best answer this by holding up a mirror. Here are some suggestions for making the most of your own death:

- Make your mind and body work well.
- Recall all your outstanding commitments and either discard or honour them.
- Take a holiday.
- Tell your loved ones that you love them.
- Do those things that you have always wanted to do.
- Be happy.
- Create.
- Make peace with yourself.
- Fall in love with yourself.

Use your imagination to prepare for and participate in your death. Although you don't know when it is coming, that is no excuse not to be ready. When it does come, be happy and adventurous. You only get to die once — make the most of it.

"I'm glad I came,
But all the same,
I must be going..."

-G. Marx □

* "Her"—why not?

Sioux Lookout

Doreen Yee 8T2

The University of Toronto offers to fourth year clinical clerks one month selectives and electives at the Sioux Lookout Zone Hospital. It is an excellent opportunity for students to observe first-hand the practice of medicine in a remote area, as well as learn about Canada's native people and their culture. Usually, three weeks are spent at the Zone Hospital working with the staff, and one week is spent in one of the nursing stations working with the outpost nurses. It is here that one is able to see the living conditions and lifestyles of the Indians.

I went up to Sioux Lookout in August 1981, spent three weeks at the hospital and one week at the Big Trout Lake nursing station. It was a valuable experience because I learned, not so much academic medicine, but how modern medicine must be tailored to the needs of the local residents in remote areas where medical personnel and facilities are limited. The following is an account of the development of the Sioux Lookout Zone Health Project, its organization and modus operandi, and my impressions of the natives and their conditions.

Sioux Lookout Zone Health Project

The Sioux Lookout Zone is one of four principal zones in Ontario. Located in the northwestern quarter of the province, it encompasses approximately 400 square miles, of the Precambrian Shield and is covered with deicuous forests interspersed with thousands of small lakes. The Zone headquarters are found in Sioux Lookout, a small town of 3500 people situated on the CN transcontinetal line, approximately midway between Winnipeg and Thunder Bay. The population of this town is mostly Caucasian, while the outpost reservations in the northern areas are populated with about 10,000 native Indians.

Prior to 1950, the only health care the natives received was from private doctors accompanying a treaty party once a year. This lack of proper health care became apparent in 1945 when pulmonary tuberculosis became epidemic among the native population. In response, the federal government established the Zone Hospital in Sioux Lookout to care for the natives. The situation improved markedly with the discovery of the anti-tubercular drugs in the early '50's. Gradually, nursing stations were built in the more densely populated reservations, and satellites set up in the smaller ones. At present, there are seven nursing stations scattered throughout the Zone, each with some satellites. Depending on the size of the community, each station is staffed by two or three nurses, while the satellites are staffed by one or two family health aides (FHA's). These are native people trained in first aid and basic primary care, and they are the ones who decide whether a patient from the satellite should be transferred to a nursing station for further care or whether they can be cared for and observed at home.

In 1969, because of severe staffing problems, the Department of Health and National Welfare asked the faculties of medicine and dentistry at U. of T. to help out. Dr. W.H. Bain, professor of paediatrics at U of T and chief of paediatrics at the Hospital for Sick Children offered the assistance of the hospital medical staff in providing health care for the natives, and the dental department did likewise. As a result, the program now supplies the Zone hospital with dental interns, senior nursing students, family practice and paediatric residents, clinical clerks and visiting consultants in various subspecialties all year around. For example, Dr. Robert Salter periodically conducts a paediatric orthopaedic clinic at the Zone hospital. Patients requiring surgery are sent down to HSC for the proper treatment. A staff radiologist visits the hospital every month to report upon all the x-rays that have been taken. In addition, Toronto Western Hospital sends a staff physiotherapist there for two month appointments.

Sioux Lookout

Sioux Lookout is a typical small northern Ontario town situated on the northern shores of Pelican Lake. The town site was originally a campsite of the Ojibway Indians. It was so named because a mountaintop overlooking the lake served as a lookout point for the Ojibwa to detect any attacking Sioux Indians. The "savage Sioux" were long-time enemies of the peaceful Ojibwa, who had been forced to flee further and further northwest from this marauding tribe. Legend

has it that 175 years ago, this tribal feud was ended when the Ojibwa surprised their Sioux attackers and managed to eradicate the whole tribe except one small boy. An Ojibway brave had spotted the Sioux from the mountain lookout, and the tribe had quickly planned a surprise attack. Today, there are almost no Sioux left, the remaining descendants are in other tribes.

There are 2 hospitals in the town: the General Hospital, mainly for the the non-native townspeople, and the Zone Hospital for the natives. Local non-natives and natives socialize together infrequently, and segregation is quite apparent except for the occasional mixed marriage.

The town's original economy was based on the railway business since it was located on the main line of the CNR. At present though, there are no large industries in the area. The majority of the locals are engaged in small commercial businesses or are employed in various departments of the government like the Ministry of Natural Resources, and the Royal Canadian Airforce Radar Base.

Most of the local townspeople I encountered were easy-going and loved the "slow pace" of small town living. On discovering a visitor from Toronto, everyone was anxious that I should like the town. When asked about what they thought of Toronto, comments like the following were expressed:

"That big city down south? Could never keep up with the pace."

"Nice place to visit on a long weekend."

"I start to feel sick and cramped if I spend more than a couple of hours there."

"Wouldn't move there for a million bucks-wouldn't leave here for the world."

It is indeed a paradise for the "outdoor person". Fishing canoeing, hunting, sailing, cross-country skiing, and snowshoeing are no more than 20 minutes away. Other modern facilities include 2 banks, 3 grocery stores, 6 restaurants, 4 churches, a Brewers' Retail, an LCBO store, and 1 theatre.

The Zone Hospital

It is essentially a general hospital with 70 beds and 5 bassinets. These are divided into 2 wards: one for obstetrics, newborns and 'clean cases', the other for infectious cases and paediatrics. There are 2 operating rooms, one for deliveries, the other for minor surgery such as D & C's and for the visiting surgeons' use. Since there are no surgeons on staff, even minor operations such as appendectomies are sent to Dryden, which is an hour's drive away.

Other facilities include an outpatient department, a library, a dental clinic, an emergency department and an X-ray department (no, there is not a CT scanner...yet!). The X-ray staff consists of one technician who is on call 24 hours a day, 7 days a week! Laboratory services include haematology, biochemistry, microbiology and a blood bank. More exotic lab tests (such as serum a-1-antitrypsin) are sent off to Thunder Bay.

The Telemedicine Project serves as a "long-distance consulting service" and teaching aid. Via telephone hookup, verbal and visual communication is possible between the Zone hospital, HSC and Sunnybrook Medical Center. Radiographs with equivocal findings may be transmitted to one of these centers for a radiologist's interpretation. "Telemedicine Rounds" or teaching rounds are held on the system once or twice a week.

Coordination of transportation is of the utmost importance in this health system. Patients are constantly being transported to and from the nursing stations to the Zone hospital, and to and from the hospital to Winnipeg or Thunder Bay in "Medi-Vacs" (standing for Medical Evacuation). Recently, the introduction of 2 air ambulances, called the "Bandage 5's" has greatly aided in the rapid transport of seriously ill patients to the hospital.

Adjacent to the hospital is a 36-bed hostel which provides room and board for natives in transit. It is used by discharged patients awaiting flights home, by expectant mothers awaiting delivery (all pre-natals are sent down to the hospital at 37 weeks), and by mothers escorting their sick children.

Also on the hospital grounds are assorted accomodations for permanent and visiting medical personnel. Twenty-four junior one-bedroom apartments are available in the Nurses' Residence. There are also housetrailers, bungalows and pre-fab houses for staff use. At present, the permanent medical staff consists of 5 GP's and an internist.

Nursing Stations

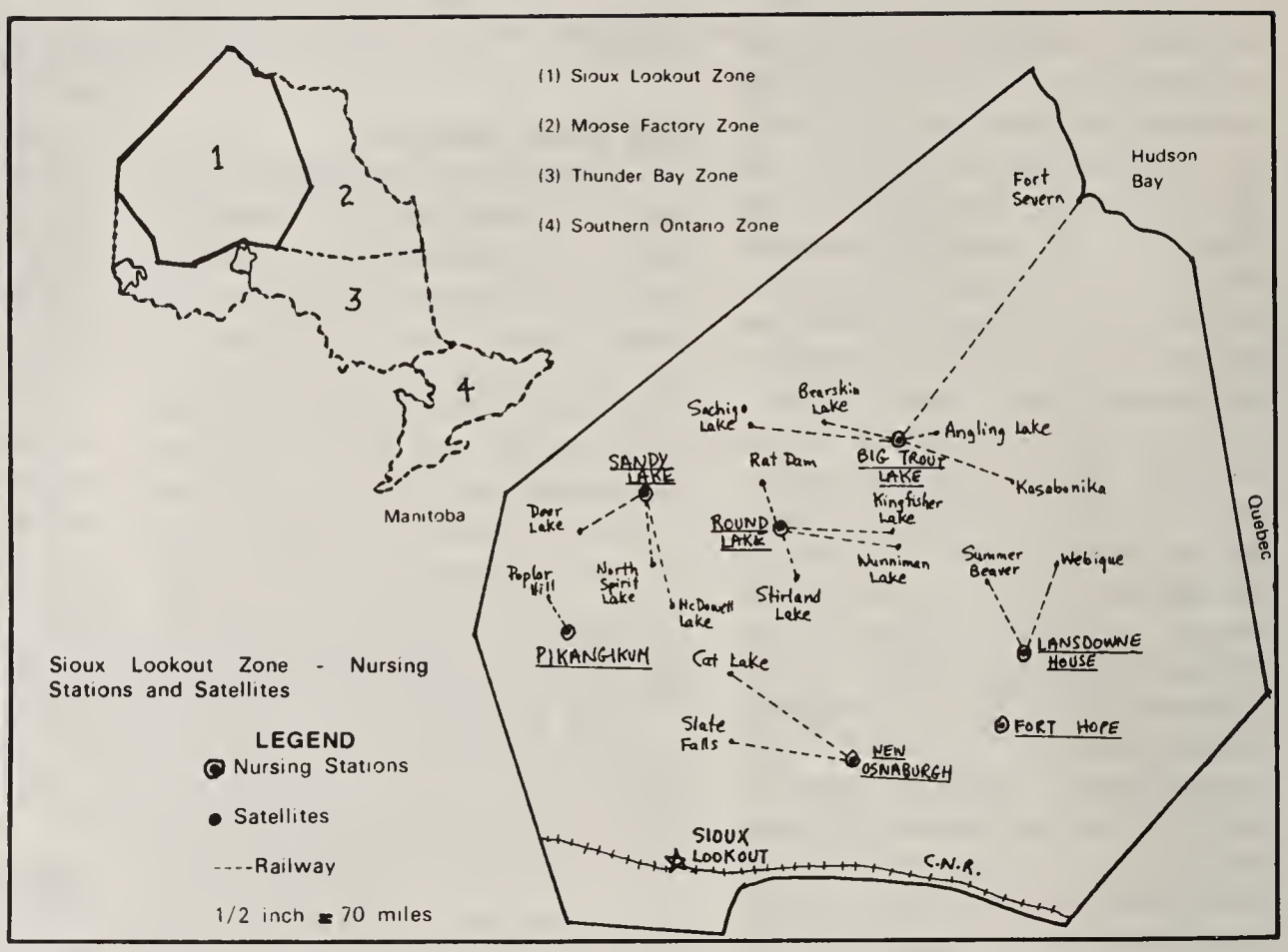
Presently, there are 7 nursing stations, each of them found in the more densely populated reserves. Each station consists of a clinic area with two or three examining rooms, x-ray facilities, 4 beds and 2 bassinets. The nurses' quarters are spacious and comfortable and are usually attached to the clinic area.

Each station is staffed by 3 nurses, at least one of whom is qualified in midwifery. In addition to nurses, local natives are hired to perform clerical and maintenance duties. GP's from the Zone Hospital also make monthly visits to the stations.

The duties of an "outpost nurse practitioner" are numerous and diverse. Services include weekly medical, pre-natal, and well-baby clinics, home visits to those unable to come to the station, and bi-monthly visits to the satellite areas. The nurses deliver babies, suture cuts, and cast uncomplicated fractures. They prescribe everything from digoxin and the "pill", to antibiotics and Kwellada. They often escort sick patients from the satellites or Sioux Lookout by air in the most treacherous weather conditions. Rather a hectic and hazardous existence one might think, but the majority of these women love the

Big Trout Lake nursing station

Sioux Lookout Zone Hospital, Medical Director's house on the left



autonomy and would find it very difficult to return to the conventional duties of a nurse in a hospital.

The turnover rate among outpost nurses is high, but for many nurses it is a good opportunity to experience the Canadian north and its people, and to make some money. The Federal Government pays for all of their living expenses, the salaries and isolation pay are reasonable, and perhaps most importantly, there are limited ways in which one can spend money.

Reserves

My first reaction on arriving at Big Trout Lake was: "How could anyone live in those little cabins all year round?" To add to my dismay, I was informed that these houses did not have running water. (How would you like to run to the outhouse in the middle of the night when it is-40^{0}F?) Cabins are generally heated by wood stoves all winter, and the heat is adequate. Most houses do, however, have electricity and many families own color TV'S, stereos and even electronic computerized games!

The lack of running water decreases the degree of hygiene considerably. Furthermore, the native population is not very informed concerning proper hygiene. Children frequently have infected insect bites are after playing outside for a whole day. Bacterial infections of every sort are rampant: cellulitis, impetigo, pneumonia, meningitis, Strep throats, and in particular, pulmonary TB. All native newborns in the Zone are routinely immunized with BCG vaccine at present. Strep throats in kids are quite common, in fact, there seemed to be an epidemic of post-strep glomerulonephritis the month I was there.

The estimated population of the Zone is about 10,000. Two nursing stations, Big Trout Lake and Sandy Lake, each have about 2000 people, while the others have approximately 1000 each. Cree Indians are generally found in the northern areas of the Zone, and the Ojibwa in the south. Though the language is different, customs and lifestyles of these 2 tribes are similar.

Among the native peoples unemployment is and will likely continue to be a major problem. Most reserves have schooling facilities with classes to the end of elementary school. Further education has to be pursued in Sioux Lookout. For most native youths, this means they get no secondary school education since it would mean leaving their homes and families for the first time to go to a strange town at age 11. The idleness of the young has led to numerous problems, one of which is gasoline sniffing among the young which sometimes results in acute and chronic lead toxicity. Alcohol and street drugs are strictly forbidden on most reserves, but gasoline is readily available. Having not much else to do, teenagers have turned to sniffing gasoline for kicks. Sniffing it brings on an almost immediate, transient "high", followed by nausea and vomiting. On some reserves like Pikangikum, this addiction has lead to many crimes. A detoxification program is now available in Winnipeg, but what these people really need is a change of environment and some sort of work and employment to give themselves a purpose and a role in life. In essence, the sad truth is that the potential and future of our native population is being wasted because "they have nothing to do after age 11". This will definitely become an enormous problem for our Ministry of Indian Affairs in the near future.□

Bench, Scalpel or Stethoscope?

Paul Boughen 8T3

The Summer Scholarship Program has been in existence for approximately 10 years and involves the distribution of scholarship funds from several sources. For the 1981 program (this past summer), the application deadline was February 1, and successful applicants were notified by mid March.

Students applying for scholarships may either select a "catalogue" project from the booklet of such projects published each spring, or they may arrange and organize a program of their own. In either case, all applications are considered and graded according to the qualities of the project. Participants in the program receive $2000.00 for a 12 week period during the summer.

This past summer, there were nine sources of funding:
James H. Cumming - 1 scholarship awarded
Bradshaw Errington - 3 scholarships awarded
Mary Cassidy Fund - 4 scholarships awarded
Lela Parlow Cancer, Research Fellowship - 1 scholarship awarded
Ciba Geigy Summer Scholarship
Mary Cassidy Fund - 1 scholarship awarded
Ontario Heart Foundation - 3 scholarships awarded
Medical Research Council - 19 scholarships awarded
P.S.I. Foundation - 51 scholarships awarded
Medical Alumni Association - 15 scholarships awarded

One requirement of the program is that at the completion of the summer, students must write a 600 word report on their activities in their project. The reports are submitted to the Awards Officer who co-ordinates the program. This brief survey of the program is based on several of these reports.

Summers and Elective Time

Within a short time of entering medical school, most students are faced with making decisions about how to spend their elective, summer and (in fourth year) selective periods. The choices of how to spend one's time seem to be the following:

a) "to do 'non-medical' activities",

b) "to further explore areas of study exposed to in medical courses",

c) "to explore one or more areas of medicine in order to decide about a career choice",

d) "to gain expertise in a decided career goal".

Research and Clinical Medicine

This past summer, 97 students were involved in the program. Of these, approximately two-thirds were second year students (8T3) and one-third were first year students (8T4). Some of the sources of funds are restricted to either clinical or research projects. For example, the P.S.I. Foundation awards are directed toward clinical programs only and the Medical Research Council gives monies only for research application. As for the location of the work, the majority (approximately 70%) of projects were completed in Toronto, but those based outside the city included many Ontario towns and cities, the coastal provinces of Canada, and even some centres outside the country, namely Australia and Taiwan.

Students were involved in an extremely wide range of projects. The clinically oriented projects involved anything from anesthesiology to urology. The supervisors were unamimously reported to be "eager to teach" and willing to let the students "get their hands dirty". This meant that the students were permitted to take histories, do physicals, decide upon management, write orders, and in some cases institute therapy. Although all of this was done under supervision, those involved reported that they felt a certain amount of responsibility was placed upon them, thus enabling them to learn both about clinical medicine and the *art* of practising medicine. Inevitably, clinical skills were improved and clinical judgement and diagnostic abilities were refined. Many of the out-of-town scholarships were spent in family practice settings, and invariably the students reported gaining a sense of good doctor-patient relationships and a reinforcement of the motto - "the patient comes first".

The scholarships awarded for research work, were virtually all based in Toronto, either in the Medical Sciences Building or in the various research wings of the teaching hospitals. Students were involved in bench work (e.g. studying the effect of LDL on endothelial cell spreading), in statistical research (e.g. a survey of

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Dr. R. D. Henderson, Toronto
Dr. Robert C. Hicks, Toronto
Dr. F. Marguerite Hill, Toronto
Dr. M. A. Hutcheon, Toronto
Dr. Aden C. Irwin, Halifax
Dr. Mary V. Jackson, Toronto
Dr. D. P. Jones, Toronto
Dr. James H Kerr, Toronto
Dr. E. C. Keystone, Toronto
Dr. J. S. Keystone, Toronto
Dr. H. King, Midland
Dr. Cecilia Lamenza, Toronto
Dr. Bernard Langer, Toronto
Dr. Eugene LeBlanc, Toronto
Dr. Jack R. Leitch, Toronto
Dr. Daniel Levy, Hamilton
Dr. Kenneth E. Livingston, Toronto
Dr. E. Llewellyn-Thomas, Toronto
Dr. L.F.W. Loach, Toronto
Dr. Frederick Lowy, Toronto
Dr. John R. Lundon, Toronto
Dr. Ross C. Mackay, Toronto
Dr. Robert Mackenzie, Toronto
Dr. Ian Macnab, Toronto
Dr. C. Mallin, Toronto
Dr. J.T. Marotta, Don Mills
Dr. A. Bruce Marshall, St. Marys
Dr. Benj. Massouda, Downsview
Dr. Rebeka Moscarello, Toronto
Dr. James F. Murray, Don Mills
Dr. R. W. McCallum, Agincourt
Dr. Raymond M. KcKeown, Oregon
Dr. F. R. Papsin, Toronto
Dr. G. A. Pengelly, Willowdale
Dr. Richard G. Perrin, Toronto
Dr. Mel. E. Petersiel, Scarborough
Dr. Burns Plewes, Toronto
Dr. C. P. Rance, Toronto
Dr. W. J. Reynolds, Toronto
Dr. Alexander Roberts, Toronto
Dr. Robert B. Salter, Toronto
Dr. E. A. Sellers, Toronto
Dr. Isaac H. Shleser, Toronto
Dr. Jack Shuber, Toronto
Dr. Ruth Sky, Toronto
Dr. A. & J. Smith, Mississauga
Dr. D. A. Stewart, Toronto
Dr. R. M. Stone, Toronto
Dr. M. Tile, Toronto
Dr. Joan M. Vale, Toronto
Dr. Daniel G. Vaughan, California
Dr. Paul G. Walfish, Toronto
Dr. Gary Webb, Toronto
Dr. K. Weber, Toronto
Dr. Douglas R. Wilson, Toronto
Dr. J. K. Wilson, Toronto
Dr. George E. Wodehouse, Toronto
Dr. M. S. Wolochow, Vancouver
Dr. C. R. Woolf, Toronto
Dr. J. R. Wright, Toronto

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the correlation between gastroscopy and upper GI series findings), and some of both types of investigation. Some research work was mixed with clinical experience as well, although this was rare. The vast majority of the research projects ended with the writing of a paper on the results of the work, and many of these papers are to be published. Many students enjoyed the exposure to laboratory work and for some the experience "reinforced an interest in medical research as a career."

SUMMARY

All the students involved in the program voiced an enthusiastic plea for the continuation of the program in the future. They unanimously felt it provided a good learning experience and hoped that it will aid them in their future decision regarding career choices.□